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# PATHOGENESIS OF LEFT BUNDLE BRANCH BLOCK

BY

HÅKON RASMUSSEN AND TORJUS MOE

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Received February 21, 1947

In 1909 Eppinger and Rothberger showed that characteristic changes in the electrocardiogram arose after section of the right and left branches of the bundle of His in dogs, and the following year these experimental results were applied to human pathology, so that bundle branch block became a clinical concept. Since that time the electrocardiogram originally designated right branch block has come to be regarded as indicative of a left-sided lesion, and vice versa, and this new conception must be considered as well-founded (Rasmussen, 1942). Apart from this correction, current opinion holds as in 1910, that the bundle branch block cardiogram is an indication of local damage to the right or left branch of the bundle of His. However, in the last ten years especially, the great similarity between the so-called *electrocardiogram of left ventricular hypertrophy* and that of left bundle branch block has often been emphasized, and it has been suggested that the latter, like the former, may be due to great enlargement of the left ventricle, without any local lesion of the branch being present.

In an investigation of the electrocardiogram in essential hypertension, Rasmussen and Thingstad (1939) concluded that great enlargement of the left ventricle was the common cause of the left bundle branch block cardiogram. In cases of hypertension with gross cardiac enlargement they found the left bundle branch block cardiogram as a sequence of the *left ventricular hypertrophy curve*. This view has been confirmed by further investigations. In a series of 100 patients with aortic insufficiency, a similar concordance between the type of cardiogram and cardiac enlargement was found as in hypertension (Rasmussen, 1944). By producing dilatation of the right and of the left heart in experiments on dogs it was possible to produce typical bundle branch block electrocardiograms (Rasmussen, 1942). On re-examination of a group of hypertensive patients, it was found that the cardiogram of left ventricular hypertrophy and of left bundle branch block developed gradually, *pari passu* with increase in the size of the heart (Rasmussen and Bøe, 1945). It was also shown that as the cardiogram changed from a normal pattern to that of left hypertrophy, the duration of QRS increased so that the curve reached the left bundle branch block type, which is characterized by a QRS of at least 0·12 sec.

Master and his associates (1940) investigated 100 patients with either left or right bundle branch block, and some with complete A-V block, and concluded that " chronic bundle branch block, in most instances, is the result of an increase in the size of the heart and of myocardial damage, with diffuse involvement of the bundle branch system." If enlargement of the left heart, by retarding the impulse to that side, can produce in the electrocardiogram a left ventricular hypertrophy curve or a left bundle branch block, is it hypertrophy or dilatation that is the essential factor? Further, it may be asked whether retardation of the impulse is due simply and solely to lengthening of its path, or to damage to the subendocardial network resulting from increased strain or distension? While for the present we may leave the latter question unanswered, we think there are grounds for believing that dilatation of one half of the heart is a decisive factor. This view is based partly upon the above-mentioned experiments relating to dilatation of the right or left heart, partly upon the well-known phenomenon that a pulmonary embolism not infrequently produces a right bundle branch block curve, presumably due to dilatation of the right heart, and also partly upon clinical observations.

We may illustrate this point of view by a few examples. Fig. 1 shows radiograms of the heart of a patient with hypertension, taken before and after sympathectomy, with an interval of about two

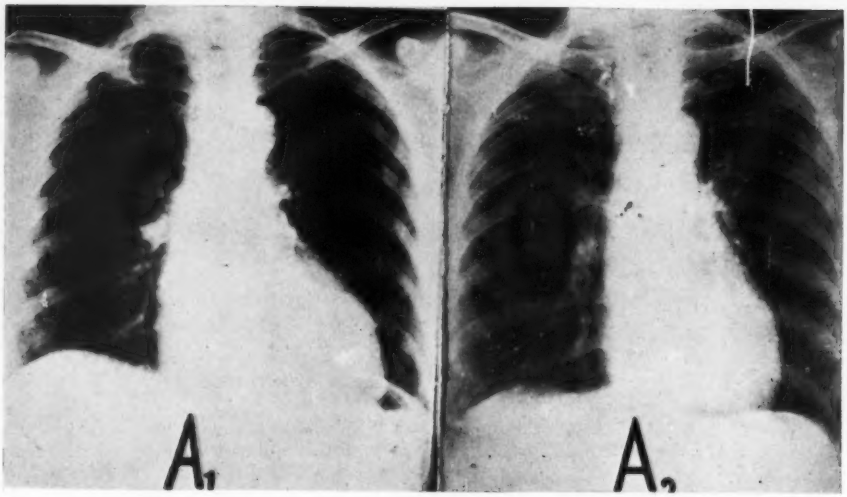



FIG. 1.—Telecardiograms from case of hypertension. (A1) before, and (A2), two months later, after sympathectomy.

months between the photographs. After operation the size of the heart is seen to have decreased considerably. The blood pressure before operation was 240/130; afterwards 120/75 mm. Fig. 2 shows the cardiograms from this case, before and after operation. Coincident with a decrease in the size of the heart the typical left ventricular hypertrophy curve has changed to a normal pattern. It seems probable that we are here concerned with dilatation which has subsided and not with hypertrophy. Fig. 3 and 4 show cardiograms and radiograms taken at an interval of 7 months from a woman

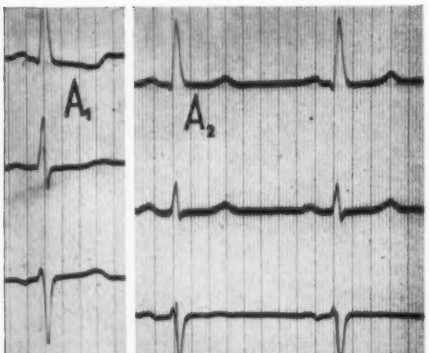
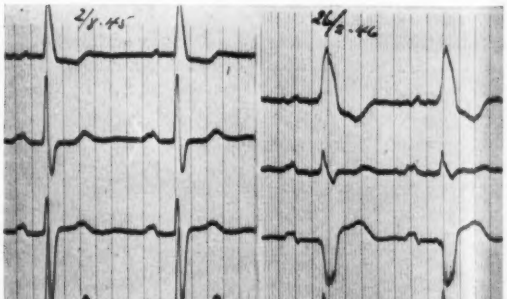
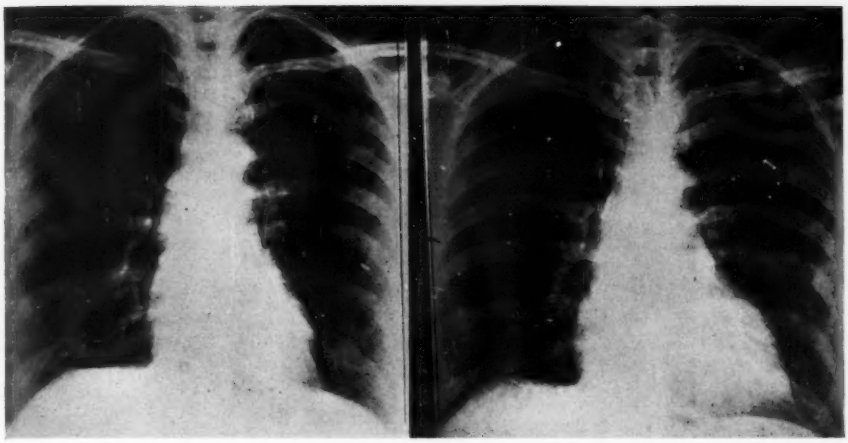



FIG. 2.—Electrocardiograms from same case as Fig. 1. (A1) before sympathectomy shows "left ventricular hypertrophy curve." (A2), two months later, after sympathectomy, showing return to normal curve.



A B

FIG. 3.—Electrocardiograms from case of aortic stenosis (A) showing "left ventricular hypertrophy curve"; (B) 6 months later, showing a left bundle branch block curve.



A B

FIG. 4.—Telecardiograms from same case as Fig. 3, showing (A) normal sized heart, and (B) 6 months later, considerable enlargement to the left.

aged 57 with aortic stenosis. The cardiogram has changed from a left ventricular hypertrophy curve to a left bundle branch block curve, coincident with a considerable increase in size of the heart, seen in the radiograms. In this case also, it seems reasonable to suppose that the enlargement occurring in the course of seven months is due to dilatation rather than to hypertrophy.

We have investigated a series of 100 patients with permanent left bundle branch block cardiograms, assembled from the Rikshospital and from Ullevål Hospital. From the clinical data, radiograms and autopsies in these cases we have attempted to determine how often left bundle branch block is associated with heart diseases causing enlargement of the left ventricle and, further, how often conditions occur that may be supposed to produce a local lesion of the branch.

The types of left bundle branch block curves found in our cases are shown in Fig. 5. The first type, with high voltage, and discordant and diphasic ventricular complexes, was found in 78 patients, the characteristic præcordial leads are also shown. The second type, where Q or S is a prominent feature, was found in 5 patients. The third type, with lower voltage, although higher than 0·5 mv., sometimes with diphasic sometimes with monophasic ventricular complexes, as in the figure, was seen in 17 patients. We have omitted from the series cases of intermittent and transient bundle branch block, the Wolff-Parkinson-White type, and bundle branch block associated with complete A-V block. The duration of QRS in our cases is given in Fig. 6. We have taken 0·12 sec. as the lower limit of QRS in bundle branch block, and the upper limit in our cases was 0·2 sec. Fourteen patients had auricular fibrillation, two auricular flutter, and six incomplete and partial A-V block.

TABLE I

AGE DISTRIBUTION IN 100 PATIENTS WITH LEFT B. B. BLOCK ELECTROCARDIOGRAM

| Age | Men | Women | Total |
|---|---|---|---|
| years | | | |
| 20–29 | 1 | 0 | 1 |
| 30–39 | 0 | 1 | 1 |
| 40–49 | 2 | 1 | 3 |
| 50–59 | 6 | 9 | 15 |
| 60–69 | 23 | 14 | 37 |
| 70–79 | 17 | 15 | 32 |
| 80–89 | 4 | 5 | 9 |
| 90–99 | 1 | 1 | 2 |
| Total | 54 | 46 | 100 |

The age distribution of our cases is shown in Table I. The youngest was 25, the oldest 91 years of age. Seventy-seven patients had dyspnœa on exertion, 31 angina pectoris on exertion, 18 anginal attacks at rest, and 17 had congestive heart failure. Nocturnal dyspnœa, sometimes with pulmonary œdema, was noted in 41 cases, indicating a very high frequency of left ventricular failure. In 5 or 6 cases, cardiac symptoms were absent or slight, the

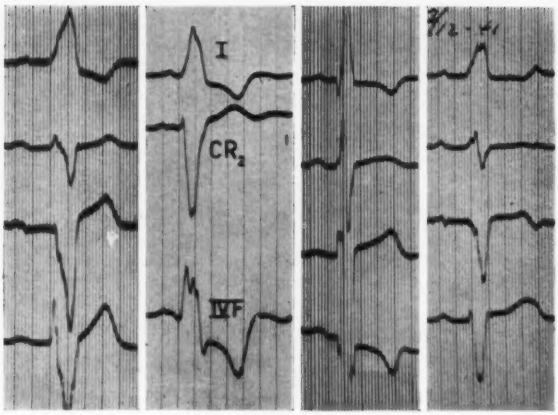



FIG. 5.—Types of left bundle branch block curve encountered. (A) The most common type and (B) typical præcordial leads; (C) rarer type with Q wave; (D) rare type with positive T wave in lead I and without high voltage. (Time marker 0·1 and 0·02 second.)

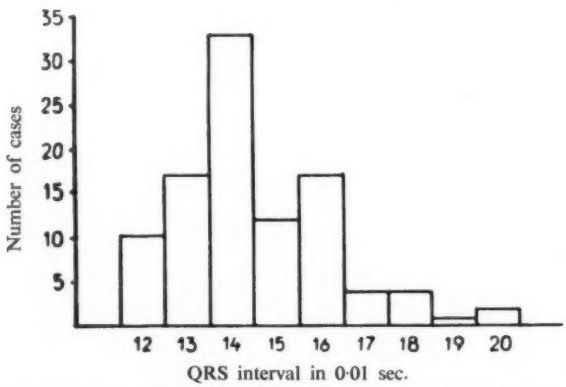



FIG. 6.—Distribution curve of duration of QRS deflections in left bundle branch block cases.

bundle branch block cardiogram being here an accidental finding in patients with other diseases (melanosarcoma, cancer of the stomach, etc.).

Table II gives the cardiac diagnoses, based upon clinical findings and autopsies, and the size of the heart in the different groups. In classifying these cases, difficulty arose mainly in regard to the hypertensive and arteriosclerotic groups. We believe that the incidence of hypertension has been underestimated, as past hypertension could not be determined from the case records. In the arteriosclerotic group have been placed patients of relatively advanced age, with moderately raised or normal blood pressure, with moderate or no enlargement of the heart, and those with signs of severe arteriosclerosis. The group with uncertain ætiology includes four patients with systolic and one with diastolic hypertension, one with syphilis, one with thyrotoxicosis, one with leukæmia, and one with pulmonary embolism.

The size of the heart, given in Table II, was assessed partly from teleradiograms and partly at

TABLE II

CLINICAL DIAGNOSIS OF HEART DISEASE (PARTLY BASED ON AUTOPSIES) AND CARDIAC ENLARGEMENT IN 100 PATIENTS WITH LEFT B.B. BLOCK

| Heart Disease | Number of cases | Cardiac Enlargement: Gross | Medium severe | Slight and moderate | Not enlarged | No roentgenogram or autopsy |
|---|---|---|---|---|---|---|
| Aortic stenosis .. .. | 13 | 7 | 3 | 2 | 0 | 1 |
| Aortic insufficiency (luetic aortitis 3) .. .. .. | 6 | 5 | 1 | 0 | 0 | — |
| Hypertensive .. .. .. | 39 | 19 | 7 | 7 | 1 | 5 |
| Probable hypertensive .. | 3 | — | 1 | 1 | 1 | — |
| Renal disease with hypertension | 6 | 2 | 2 | 2 | — | — |
| Hypertensive with myocardial infarction .. .. .. | 9 | 6 | 1 | 0 | 1 | 1 |
| "Left-sided" heart disease .. | 76 | — | — | — | — | — |
| Myocardial infarction .. .. | 3 | 1 | 0 | 0 | 1 | 1 |
| Arteriosclerotic .. .. | 6 | 1 | 0 | 3 | 1 | 1 |
| Uncertain or unclassifiable .. | 14 | 4 | 4 | 4 | 1 | 1 |
| No heart disease (melanosarcoma) .. .. .. | 1 | 0 | 0 | 0 | 1 | — |
| Total .. | 100 | 45 | 19 | 19 | 7 | 10 |

autopsy. The grouping was done by making a rough estimate of the size of the heart in two planes, and comparing it with the cardiothoracic index. This rough estimate is thought to be more valuable than the cardiothoracic index alone. Hearts weighing up to 350 grams are regarded as normal, those up to 500 grams as slightly enlarged, from 500 to 599 grams as moderately enlarged, and those weighing more than 600 grams as greatly enlarged.

It is evident from Table II that conditions involving increased work for the left ventricle greatly predominate in our material. Altogether, such conditions were present in 67 cases. In the combined group, hypertension with cardiac infarction, left bundle branch block was certainly present before the occurrence of infarction in 3 cases and in only 4 of the 9 cases in this group could the bundle branch block be attributed to the infarction. Including the remaining 5 cases from this group, there were 72 cases in all in which an affection of the left heart was regarded as the dominant pathogenic factor in the causation of bundle branch block. In addition, there were several cases of slight hypertension in the arteriosclerotic group and the group of uncertain ætiology.

The possibility of local damage to the branch exists in the three cases with infarction, in the six with arteriosclerotic heart disease without certain infarction, in four of the nine with hypertension and infarction, and possibly in the case with metastatic melanosarcoma, even though myocardial metastases were not found at autopsy, that is to say in 14 cases altogether.

Having thus established a high incidence of left ventricular heart disease in our cases, namely over 70 per cent, we may now consider how often the left heart was sufficiently enlarged to account for the left bundle branch block. Reference to Table II shows that out of 90 cases in which X-ray or autopsy data were available, there were 45 with gross and 19 with moderately severe cardiac enlargement, or altogether 64 cases (71 per cent) with enlargement of a degree that we may suppose sufficient to explain the bundle branch block.

Both radiographic and post-mortem examination revealed enlargement which affected the left heart predominantly, and in only 4 or 5 cases was any considerable degree of co-existent right-sided enlargement found. It may be noted that a heart may enlarge, as proved by serial radiographs, and yet remain within the upper normal limit of size. Here the electrocardiogram may often give better information in the form of the characteristic picture of left ventricular hypertrophy.

Fig. 7 shows how 71 radiographically examined hearts are distributed in relation to the cardiothoracic index. While 7 cases (+5) show normal values, 43 of the 71, or 60 per cent have a lower index than 1·80. The average cardiothoracic index is 1·74. If we compare this graph for the size of the heart in cases of left bundle branch block with that drawn

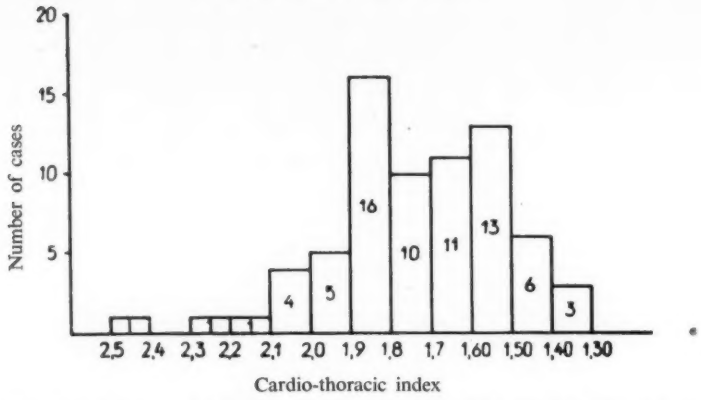



FIG. 7.—Distribution curve of cardiothoracic indices in 71 patients with left bundle branch block.

by Ottar Müller (1942) for the left ventricular hypertrophy curves (see Fig. 8), we see that there is some displacement to the right, i.e. towards the larger hearts, in cases of left bundle branch block, otherwise the two graphs are very similar. Of the left bundle branch block patients 60 per cent had an index below 1·80, compared with 48 per cent of Müller's cases with left ventricular hypertrophy curves. We may add that many of the largest hearts found post-mortem in cases of left bundle branch block were not radiographically examined. The cardiothoracic index is an insufficiently accurate standard of heart size to permit more elaborate statistical analysis.

The weight of the heart in 31 cases examined post-mortem is given in Table III, which shows that we are dealing with extremely large hearts, 17 of them weighing more than 600 grams and the average being 652 grams. The largest heart, weighing 1780 grams, was from a 25-year-old man with aortic insufficiency and stenosis, as well as slight mitral disease and great thickening of the pericardium. White states in his textbook that the largest heart known to have existed weighed 1755 grams (Smith,

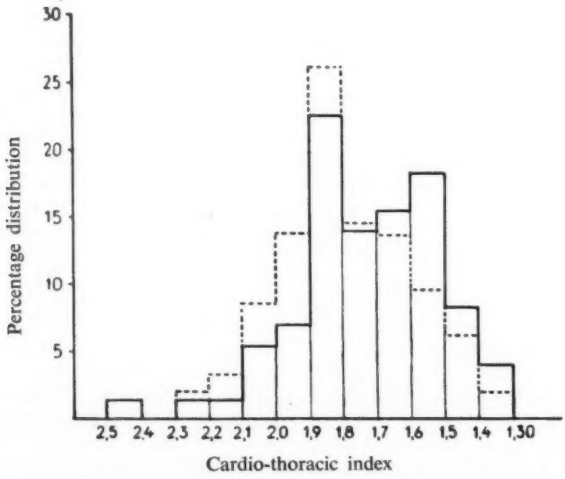



FIG. 8.—Percentage distribution of cardiothoracic indices in left bundle branch block (continuous line) compared with those in left ventricular hypertrophy (dotted line).

TABLE III

HEART WEIGHTS IN AUTOPSIES FROM 31 PATIENTS WITH LEFT B. B. BLOCK

| Heart weight | Number of cases | Remarks |
|---|---|---|
| 350 g. and smaller | 2 | 320 and 350 g. |
| 351–449 g. .. | 3 | |
| 450–499 g. .. | 5 | |
| 500–599 g. .. | 4 | |
| 600–799 g. .. | 11 | |
| 800–999 g. .. | 4 | |
| 1000 g. and more | 2 | 1030 and 1780 g. |
| Total | 31 | |

1855), so that we have in this series the *largest heart in the world*, but as in most of such huge hearts, there was great thickening of the pericardium. For two of the hearts the weight was reckoned to be normal.

In identifying the electrocardiogram of left ventricular hypertrophy, weighing the heart (and particularly the two ventricular muscles separately) has played a certain role (Lewis, 1913–14; Hermann and Wilson, 1921). We have given reasons for assuming that dilatation is of greater importance than hypertrophy of the muscle, and one of our two cases with a heart of normal weight may be cited to illustrate this point. A woman, aged 58, who had a heart affection of uncertain ætiology with angina pectoris, increased basal metabolism and leukæmia, was found six months before death to have a considerably enlarged heart in radiographs, the heart volume being estimated at 985 ml., yet at autopsy the heart was found to weigh only 320 grams.

It is obvious that many of the hearts from patients in these age groups will show arteriosclerotic changes in the coronary vessels. The significance of reported post-mortem findings, such as "some sclerosis, no stricture," "highly sclerotic, no distinct obstruction," "extreme sclerosis," etc., cannot be judged in relation to the organic or functional disturbances that may be occasioned by a reduced blood supply to the heart muscle.

## SUMMARY AND CONCLUSIONS

In the course of previous investigations relating to the electrocardiogram in hypertension and in aortic incompetence, we reached the conclusion that the left bundle branch block cardiogram might be caused by gross enlargement of the left ventricle apart from any local lesion of the bundle branch.

In the present investigation we have sought to test this hypothesis further by an analysis of the clinical, radiological, and necropsy findings in a series of 100 cases, presenting permanent left bundle branch block in the cardiogram. It was found that diseases affecting the left ventricle, such as hypertension and aortic valvular disease, predominated in this material, occurring in 72 per cent of cases, and that a considerable degree of left ventricular enlargement occurred with about the same frequency. In 14 cases there were reasons for presuming the existence of local damage to the left branch of the bundle, of which the most important cause was probably cardiac infarction involving the ventricular septum.

Our investigation suggests that the left bundle branch block electrocardiogram is five times more often due to enlargement of the left heart than to a local lesion of the left branch of the bundle. In this connection dilatation is deemed to be more important than hypertrophy.

We have reason to believe that the *left ventricular hypertrophy curve* and the *left bundle branch block curve* represent different degrees of retarded conduction to the left heart, and that, therefore, no sharp distinction between them is necessary. Both imply, in most but not in all cases, a considerable degree of left ventricular enlargement, and both are found in association with diseases that affect the left heart. The terminology at present applied to these electrocardiograms is inadequate and the comprehensive term *electrocardiogram of left-sided retardation* might with advantage be employed to designate all patterns of electrocardiogram that indicate retarded conduction to the left heart, of which the bundle branch block type represents the most extreme grade.

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# EROSION OF RIBS IN COARCTATION OF THE AORTA

## A NOTE ON THE HISTORY OF A PATHOGNOMIC SIGN

BY

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The reader of Bramwell's excellent study of coarctation of the aorta (1947) will have obtained a somewhat incomplete and faulty impression of the place of rib erosion in the diagnosis and in the medical history of that anomaly, for he is referred to " Roesler's classical paper " of 1928, and " similar observations " of Railsbach and Dock published " the following year." In an earlier paper describing the study of the distribution of notching of the ribs, Bramwell and Jones (1941) had not dealt with the historical side.

Erosion of the ribs due to coarctation was first emphasized by English clinicians—Craigie (1841) and Walshe (1873)—as a regularly observed anatomical lesion. It was completely overlooked by early radiologists, and in Roesler's long paper on coarctation (1928) the presence of erosions in one case was noted in passing. As Roesler did not refer to the reports of Meckel (1827) and Jordan (1830), or to the English literature, he failed to draw attention to rib erosion as an important point in diagnosis. Rib erosion is not referred to in the title of the paper, which did not attract the attention of radiologists. This paper was in the December 1928 issue of *Wiener Archiv für Innere Medizin*, which, like many Austrian publications, came out weeks after its date of issue.

The paper of Railsbach and Dock (1929) entitled *Erosion of the Ribs in Stenosis of the Isthmus (Coarctation) of the Aorta* appeared in *Radiology*, January 1929, and was in the hands of subscribers during that month. Since most of these readers had in their files cases showing this lesion, but undiagnosed, and cases of known coarctation in which the erosions had been missed, it is safe to say more erosions were first correlated with the ætiological factor in the first three months of 1929 than in any similar period in history. Awakening of interest in coarctation among radiologists dates from that article. As the authors were familiar with the nineteenth century literature, they emphasized that these erosions were pathognomonic, and they minimized the diagnostic value of study of the cardiac and aortic silhouettes. With this, all subsequent authors have agreed. Roesler's paper is a classic on the cardiac silhouette in coarctation, but later authors failed to corroborate his views on its diagnostic value. Railsbach and Dock's paper, which appeared simultaneously, stressed erosion of the ribs, summarized older anatomical knowledge, reproduced Meckel's original picture of rib erosion (1827) and has been confirmed by all subsequent students. In routine chest films more cases of coarctation are detected by rib notching than by all other methods of physical examination put together. No cases are diagnosed by the cardiac silhouette in such routine films. Many cases, overlooked by physicians who recorded elevated blood pressure, have been picked up from rib erosions seen in 35 mm. films of employees or college students, or in the routine films of the American draftees and enlisted personnel.

For the English reader it may be of interest that a fact known to the great English cardiologists was confirmed and its practical value in the Roentgen era brought to the attention of radiologists by a California physician. Just as Cesalpino's fellow-countrymen ignored his discovery and description of the circulation of blood, Craigie's and Walshe's fellow-countrymen were unaware of the classic descriptions of rib erosion as a result of coarctation of the aorta, and those who spoke the same language as Meckel were not aware of Meckel's paper on this subject. Priority of discovery belongs to Meckel.

## EDITORIAL NOTE

An interesting point in connection with Meckel's original illustration of the collateral vessels in coartation of the aorta is that the erosions are shown on the upper borders of the ribs, though as we now know they actually occur only on the lower borders. Meckel's figure shows erosions on the upper borders of the third and fourth ribs on the right side, none being shown on the left side. It seems probable that the artist was responsible for this remarkable error.

Dr. Dock refers to Crighton Bramwell's paper on coarctation, and it is interesting to find that the only contemporary reference to Walshe's statement that the dilated collateral vessels might wear away the ribs is in Byrom Bramwell's *Diseases of the Heart* published in 1884.

Maude Abbott (1928), in her monograph on coarctation of the aorta in the *American Heart Journal*, April 1928, published a radiograph in which erosion of the ribs is well seen, and a few pages later reproduced Meckel's original anatomical figure together with a legend in which the rib erosions are specified, yet the radiographic notches escaped her notice.

At the meeting of the Cardiac Club in 1930, I presented "a case of coarctation of the aorta showing Roesler's sign." This was a patient under the care of Dr. G. E. Beaumont at the Middlesex Hospital in whose radiograph notching of the ribs was noticed by Dr. Maurice Weinbren in May 1929. In seeking an explanation, he came across the paper of Railsbach and Dock, to which he drew my attention. Before presenting the case, I discovered Dr. Roesler's reference to rib erosion and discussed it with him in Vienna, which explains my use of the eponym at the time.

The merit of drawing attention to the diagnostic significance of rib erosion certainly belongs to Railsbach and Dock, who first portrayed it and correlated it with previously recorded anatomical observations which had been generally overlooked. Dr. Dock emphasizes the importance of Meckel's original description, even though Meckel portrayed the rib notches incorrectly.

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# COARCTATION OF THE AORTA

## REVIEW OF TWENTY-THREE SERVICE CASES

BY

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In Maude Abbott's series of 200 autopsied cases of coarctation of the aorta published in 1928, there were only 21 cases in which the diagnosis had been made during life. Since Lewis' paper (1933), and especially since the radiological features of coarctation were described by Roesler (1928) and by Railsback and Dock (1929), the clinical diagnosis has been made far more frequently.

The purpose of this paper is to present a brief analysis of 20 cases of coarctation of the aorta who served in the recent war, that have been collected in going through the records of the Ministry of Pensions service documents. These cases have not been examined personally, but their records contain full details of pre-war occupation, examination prior to enlistment, duration and nature of service, clinical and radiological signs of coarctation found, and sometimes the after history. To these 20 cases have been added 3 who served in the 1914–18 war where the diagnosis of coarctation was made in later years. One of these has been described by Lewis (Case 2) but it has been included here because the service documents provide additional information of interest.

### Service History

*Age.* Table I shows the age when coarctation of the aorta was first diagnosed in the recent war cases. The youngest was 19 and the oldest 37: five were over 30 years of age. All were males.

*Body Build.* Table I also shows the height and weight on entry into Service, at a younger age than that recorded in column 2, when the coarctation was diagnosed. The striking feature was the good physical development and nourishment in the majority. In about half of the series the recorded weight on entry into the services was over 140 lb. Remarks such as " sturdy build," " heavily built," " powerful physique " were recorded in many cases, and all but 3 were graded fit for general service, or Grade I on entry. In the three exceptions, the lowered category was due to the condition of the feet in one (Case 11), to the eyes in another (Case 18), and only once to the heart, in Case 6, diagnosed as mitral regurgitation on grounds of a systolic murmur heard in the mitral and aortic areas; the body weight in this case was 167 lb.

It is interesting to note at this stage that in four other cases (10, 11, 12, and 19) a systolic murmur was noted at examination on entry, which was not regarded of such significance as to justify lowering the category. Case 12 was graded I after a cardiologist's report diagnosing a slight congenital defect. In five out of the twenty cases, therefore, a cardiac murmur was known to be present prior to the diagnosis of coarctation.

*Pre-Service Occupation.* It will be seen (Table I) that in only two of the series was the occupation sedentary. Of the non-sedentary occupations about half may be considered as involving heavy work. Case 5 was an athlete who played rugby football and was a quarter mile runner. Case 7 played all games well at school and played football when he joined the Navy. Case 19 was a county hockey player.

*Pre-Service history of Rheumatism.* On examination prior to entry into Service all are questioned, and the answer recorded, as to whether he or she has suffered from rheumatic fever or rheumatism in the past. In the series of 20 recent war cases there was a history of rheumatic fever, rheumatism or chorea in 4. Case 2 recorded chorea at the age of 12, with 4 months absence from school; Case 4 muscular rheumatism in adolescence; Case 5 rheumatic fever when aged 7 and a recurrence at age 10, and Case 18 rheumatic fever when aged 18. This series, however, is too small to come to any conclusion as to whether cases of coarctation of the aorta are more vulnerable than normal to rheumatic infection. In Abbott's series of 70 cases, only 8 had a history of rheumatism.

*Service prior to Cardiac Breakdown.* The majority

TABLE I

TWENTY CASES SERVING IN RECENT WAR

| Case | Age when first diagnosed | Height | Weight | Pre-Service occupation | Examination on enlistment |
|---|---|---|---|---|---|
| | | ft. in. | lb. | | |
| 1 | 24 | 5 10 | 150 | Grocer's assistant | Fit for service in any part of the world. |
| 2 | 24 | 5 4½ | 116 | Fish fryer | Physical development good. Grade I. |
| 3 | 23 | No record | | Labourer | Well developed. A.1. |
| 4 | 24 | 5 9¾ | 164 | Grocer | Sturdy build. Grade I. |
| 5 | 21 | 5 10 | 145 | Motor-driver's mate | Athletic. A.1. |
| 6 | 20 | 5 8 | 167 | Builder's labourer | Systolic murmur. Mitral regurgitation. Grade III. |
| 7 | 20 | No record | | General labourer | Large man. Grade I. |
| 8 | 33 | 5 8 | 152 | Labourer | Grade I. |
| 9 | 32 | 5 6 | 139 | Lorry driver | Grade I. |
| 10 | 25 | 5 10½ | — | Clerk | Heavily built. Systolic murmur. Grade I. |
| 11 | 35 | 5 7 | 130 | Bill-poster | Systolic murmur at apex. Grade IIa (feet). |
| 12 | 19 | 5 11 | 136 | Transport mechanic | Well developed; soft systolic murmur. Slight congenital defect. Grade I. |
| 13 | 27 | 5 4 | 120 | Labourer | Grade I. |
| 14 | 20 | 5 7 | 146 | Electrician | Powerful physique. Grade I. |
| 15 | 37 | 5 5½ | 123 | Labourer | Grade I. |
| 16 | 20 | No record | | Model-maker | A.1. |
| 17 | 24 | 5 5½ | 126 | Apprentice moulder | Fit for general service. |
| 18 | 35 | 5 7½ | 152½ | Dock labourer | Grade IIa (vision). |
| 19 | 20 | 5 9½ | 147 | Surveyor | Slight mitral murmur. Compensation good. Grade I. |
| 20 | 29 | 5 9 | 147 | Draper's assistant | Physical development good. Grade I. |

served for several years as Grade I men, doing intensive training, and subjected to all the stress and strain of war, including overseas service. Two (Cases 11 and 15) were only discovered on routine examination for service overseas after two years' in the forces. This clearly demonstrates that men with coarctation may have the capacity to withstand severe physical strain for prolonged periods without any symptoms or signs of heart failure appearing.

## CLINICAL FINDINGS

Table II gives an analysis of the clinical findings in 22 cases including 3 of the 1914–18 war (Cases 21, 22, and 23). Case 20, only diagnosed at autopsy, is not included.

*Arterial Blood Pressure.* A raised blood pressure in the upper limbs is one of the most constant signs in coarctation of the aorta. From Table II it will be seen that this was raised in every case in this series. The lowest pressure recorded was 144/90 mm. (Case 19), but this man was only 20 years of age when the coarctation was diagnosed. He was aware that he had a " leaking heart " as a lesion had been discovered on routine examination at school, when aged 15. As there were no symptoms, no restriction on activity was imposed; he played strenuous games and was a county hockey player. When examined a slight mitral murmur was detected but compensation was stated to be good and he was passed fit for general duties. After two years' service, coarctation of the aorta was diagnosed but in the absence of symptoms he was retained in Category C. He remained well for another three years until subacute bacterial endocarditis supervened and rapidly proved fatal.

Where the blood pressure is recorded separately in each arm it will be noted that there is sometimes a difference between the two readings. Usually it is lower in the left arm owing to the diminished circulation that results from the coarctation encroaching upon the orifice of the left subclavian artery. In four cases, where the blood pressure in both arms was recorded, it was lower in the left arm than in the right, and once (Case 6) it was lower in the right.

*Blood Pressure in the Legs.* In every case there was a marked lowering of the arterial pressure in the lower limbs compared with the upper, and in many there was absence of the femoral pulse. This emphasizes the importance of always palpating the femoral pulse in hypertension, especially in young people. As the result of deficient circulation to the lower limbs symptoms of intermittent claudication may occur, as happened in Case 7 four years after coarctation had been diagnosed.

TABLE II

ANALYSIS OF CLINICAL FINDINGS IN 22 CASES

| Case | Blood pressure | | Murmurs | Pulsation root of neck | Collateral circulation | Cardiac enlargement |
|---|---|---|---|---|---|---|
| | Brachial | Femoral | | | | |
| 1 | RA.185/110 LA.150/90 | 102/? | Loud, blowing systolic axilla and back | + | − | + |
| 2 | 200/140 | — | Soft systolic and blowing diastolic; loud systolic medial to scapula | + | + | − |
| 3 | 156/92 | 104/80 | Harsh loud systolic all areas and back | + | + | Slight |
| 4 | 225/120 | — | Systolic—also over internal mammary and supra scapula arteries | + | + | + |
| 5 | RA.250/160 LA.210/120 | — | Loud systolic præcordium and back | + | + | Slight |
| 6 | RA.195/110 LA.225/115 | — | Systolic apex and aortic area | − | − | + |
| 7 | RA.204/120 LA.188/110 | — | Systolic præcordium | + | + | + |
| 8 | 210/100 | — | Soft systolic apex: diastolic base | − | − | + |
| 9 | 225/115 | 175/120 | Systolic all areas | + | + | + |
| 10 | 190/100 | — | Loud apical systolic | + | + | + |
| 11 | 200/110 | 140/120 | Loud systolic præcordium | + | + | + |
| 12 | 200/110 R and L | 110/? | Soft systolic over præcordium and paravertebral region near scapula | − | − | + |
| 13 | 200/105 | — | Loud systolic over præcordium | − | − | − |
| 14 | 210/110 | — | Rough basal systolic | − | − | − |
| 15 | 230/120 R and L | 130/? | Rough basal systolic maximal pulmonary area | + | + | − |
| 16 | 168/100 R and L | ? | Blowing systolic over pulmonary area | − | − | − |
| 17 | RA.195/105 LA.185/95 | 80/2 | Harsh aortic systolic and blowing diastolic | + | + | + |
| 18 | 180/110 | — | Loud systolic all areas best heard over 3rd left space | − | − | + |
| 19 | 144/90 | — | Loud systolic all areas. Loud diastolic 2nd and 3rd right space | + | + | + |
| 21 | 190/110 | 130/? | Systolic apex and 2nd right space | + | + | + |
| 22 | 178/102 | — | Loud blowing systolic at apex conducted widely | + | + | + |
| 23 | 276/120 | 156/120 | Systolic over all areas including back | + | + | + |

*Murmurs.* The characteristic murmurs of coarctation of the aorta are systolic in time, sometimes loud and harsh in intensity, and heard all over the præcordium, often at the back in the region of the scapula, and over the dilated arteries forming the collateral circulation. Systolic murmurs were recorded in every case in this series. In 7 the murmur was heard posteriorly. Reifenstein *et al.* (1947) in reviewing 104 autopsied cases state that systolic murmurs were heard in all 60 in which mention was made of physical examination of the heart, and in 23 of them the murmur was heard posteriorly. A systolic murmur is, therefore, to be regarded as one of the most constant findings in coarctation.

In four (Cases 2, 8, 17, and 19) a diastolic murmur was also present. Reifenstein *et al.* state that a diastolic murmur is not found in uncomplicated coarctation of the aorta, but only when there is an associated aortic valvular deformity, including bacterial endocarditis or patency of the ductus arteriosus. In Case 2 of my series, the diastolic murmur was most likely due to acquired rheumatic aortic incompetence, dating from an attack of chorea when he was 12, which necessitated four months absence from school. In Case 8 the diastolic murmur was also likely due to an associated aortic regurgitation, for there was a history of rheumatism, and though the Wassermann reaction during Service was strongly positive, it was not considered to have any connection with the cardiac lesion. In Case 17 radiological examination showed calcification of the aortic arch and probably an aneurysmal dilatation, which would explain the diastolic murmur. Case 19 is the one already described which developed bacterial endocarditis, and an aortic valvular defect was probable. Con-

genital bicuspid aortic valve is a common associated lesion in coarctation of the aorta, occurring in 24 per cent of Abbott's series (1928).

*Pulsation at the root of the Neck.* Bramwell has pointed out that excessive arterial pulsation at the root of the neck should be included as one of the classical signs of coarctation of the aorta. In my series this sign was noted in 15 and not noted in 7 of the 22 cases. Once (Case 21) the association of marked visible pulsation in the suprasternal notch with a murmur in the aortic area, transmitted to the neck, led to an initial diagnosis of aortic aneurysm.

*Collateral Circulation.* Dilated collateral vessels were not observed in 9 of the 22 cases in this series, and therefore are not to be regarded as a sign that is essential for diagnosis of coarctation. Campbell and Suzman (1947) have recently described a special method of demonstrating the collateral vessels when they are not normally evident.

*Radiological Diagnosis.* Although coarctation of the aorta may be diagnosed on clinical signs alone, many more cases have been diagnosed during life since erosion or notching of the lower borders of the ribs was described by Roesler (1928) and by Railsback and Dock (1929). Notching of the ribs depends on the development of the collateral circulation inside the thorax, and is not present in every case. If there is a patent ductus arteriosus, rib notching is usually absent (Bramwell, 1947).

In the 22 cases of my series, rib erosion was recorded in 16, and was probably present in a further case (Case 6) in which coarctation of the aorta was diagnosed radiologically, though details were not given. In Case 1, although no rib erosion was detected, there was some erosion seen at either end of the left clavicle. This leaves only 4 out of 22 cases in which no erosion occurred (Cases 4, 9, 10, 21), and in these collateral vessels were present. In Bramwell's series of 26 cases rib notching was absent in 4 cases. On the other hand, 6 cases showed notching of the ribs without clinical evidence of a collateral circulation (Cases 8, 12, 13, 14, 16, and 18). It must be concluded, therefore, that rib notching is a valuable diagnostic sign of coarctation of the aorta.

Absence or smallness of the aortic knuckle also appears to be a valuable sign of coarctation of the aorta, for in only two cases (Cases 10 and 14) was the aortic knuckle not reduced in size. In Perlman's series of 13 cases, the aortic knuckle was absent in every case, and all of them showed some rib erosion.

Radiological enlargement of the heart, usually the left ventricle, was noted in 16 of 21 cases of this series. (In Case 6, details of radiological findings were not recorded.) Enlargement of the heart did not appear to have any relation to the blood pressure. In 4 of the 5 cases with no enlargement the systolic blood pressure in the arms was 200 mm. or over (Cases 2, 13, 14, and 15). Unlike essential hypertension, the high blood pressure in coarctation of the aorta does not necessarily cause cardiac enlargement, as Lewis has emphasized.

In five (Cases 1, 4, 5, 6, and 16), the diagnosis was made by the radiologist. Case 1 was at first diagnosed as mitral stenosis. In Case 4 no cardiac disability was suspected prior to a routine X-ray examination of the chest whilst convalescing from diphtheria. Case 5 was X-rayed following a collapse whilst playing rugby football. Case 6 was at first thought to be one of polycythæmia vera with hypertension. In Case 16 the chest was X-rayed on account of bronchitis and asthma.

In Case 10 there was no radiological evidence of coarctation of the aorta. This man was a clerk before he joined the Royal Air Force in July 1940. At his medical examination prior to enlistment a systolic murmur was noted but was not regarded as of any significance and he was graded I. There was no history of rheumatic fever or chorea but he was unable to play games during his last four terms at school owing to shortness of breath. In March, 1941, he was examined for fitness for a commission as a pilot and was rejected. A systolic murmur was noted at the apex and his blood pressure was 188/92 mm. The diagnosis of hyperpiesia with cardiac bruit was made. He was re-examined in October, 1941, owing to complaints of dyspnoea and attacks of pain over the heart, and his blood pressure was then 200/100 mm.; he was still Grade I. He was referred to a specialist who diagnosed mitral disease with hypertension. He continued to serve until October, 1944, when he collapsed and was found unconscious at a railway station whilst being posted. He was transferred to Middlesex Hospital where, after complete investigations, coarctation of the aorta was diagnosed, although there was no radiological evidence of it. No erosion of the ribs was present and the aortic knuckle was prominent and high. Tomograph of the aorta revealed no narrowing. The barium swallow showed no enlargement of the left auricle but the left ventricle was slightly enlarged. However, all the characteristic clinical signs of coarctation were present, namely increased blood pressure in the upper limbs (190/100 mm.), absence of femoral pulsation, marked arterial pulsation in the neck and below the outer third of the clavicle, a loud systolic murmur at the apex of the heart and a collateral circulation in the axilla and scapular regions.

Electrocardiographic examinations were recorded

in several cases but were not of diagnostic help. In Case 19 a conduction defect was shown.

*Prognosis.* Bramwell concluded that patients with coarctation of the aorta whose symptoms date from childhood are unlikely to reach the age of 30, whereas in those who are free from symptoms until 30, the outlook is much more favourable.

All of the recent war cases under discussion have reached adult life; the youngest case was aged 19 and the oldest aged 37; fifteen were under 30 years of age. As already stated, the majority were of good physical development and fit enough to be graded I. It would appear, therefore, that if symptoms do not become manifest in childhood there is no retardation of physical development or diminished fitness, and that such subjects are able to perform heavy physical work for a prolonged period without any symptoms or signs of cardiac failure, though eventually prolonged stress may cause cardiac symptoms.

Table III shows the history of the 20 recent war cases after discharge. Three deaths are recorded. Case 20 died suddenly, at the age of 29, from rupture of the aorta whilst on leave after five years' army service as a Grade I man. Neither prior to, nor during his five years' service, had there been any symptoms or signs of heart trouble. He was examined on entry, and had twice been passed fit for overseas service without the detection of any cardiac disability. The autopsy findings were as follows :—

"Very well-developed, well-nourished man.

TABLE III

HISTORY OF RECENT WAR CASES AFTER DISCHARGE

| Case | Date of diagnosis | Date of discharge | |
|---|---|---|---|
| 1 | 5.1.40 | 16.4.40 | Worked as N.A.A.F.I. manager. Developed pulmonary tuberculosis 21.10.45. October, 1947, well and working. |
| 2 | November, 1945 | 24.3.46 | Working as a fitter, only symptom slight shortness of breath on exertion. |
| 3 | 11.8.43 | 23.3.44 | Clerical work. Complained of fainting attacks. Admitted to Royal Infirmary, Edinburgh, 18.12.46 for operation. Died from cardiac failure at close of operation. |
| 4 | 3.8.43 | 11.12.43 | Resumed pre-service occupation: has occasional days off. |
| 5 | December, 1940 | 10.1.41 | 17.9.46. Worked as light vehicle driver and later as fitter. Short of breath, headache, occasional dizziness. |
| 6 | 17.2.41 | 2.4.41 | No further history: no claim for pension. |
| 7 | 15.2.42 | 31.3.42 | 15.10.46. Working general handyman in building trade. Shortness of breath on exertion: intermittent claudication. |
| 8 | 26.6.44 | 11.6.40 | Invalided from service, diagnosis myocardial degeneration. Coarctation diagnosed by medical board 26.6.44. On 13.12.46 working in grocery business. B.P. 200/120. No congestive failure. |
| 9 | 22.3.45 | 4.10.45 | No further history. |
| 10 | 3.10.44 | 21.4.45 | Working in radio trade. |
| 11 | 14.2.44 | 13.6.45 | 4.2.47. Complains of headaches, easily tired, short of breath on exertion. Works at pre-war occupation—bill poster. |
| 12 | 6.4.45 | 19.8.45 | 21.1.47. Shortness of breath, occasional pain in the chest, dizziness. Worked for three months after discharge then gave it up. |
| 13 | November, 1945 | 19.11.45 | 5.12.46. Pain across upper part of chest at night lasting half hour. Works as refuse collector for council: no loss of time. B.P. 190/120. No evidence of enlargement of left ventricle. |
| 14 | 23.6.41 | 28.3.44 | 24.1.47. Shortness of breath on exertion, feels tired. General condition good. B.P. 225/110. |
| 15 | 11.5.44 | 7.6.45 | July, 1947. Pains over præcordium. Works as lorry driver. Heart not enlarged. Visible pulsation in neck. |
| 16 | 22.12.44 | 16.3.45 | 7.10.46. Short of breath on exertion. Joiner, March, 1945 to May, 1946, then lighter work as telephone wireman. General condition good. |
| 17 | 27.8.45 | 9.1.46 | 11.3.47. Shortness of breath and dizzy attacks. Clerk, loses one day a week. B.P. 235/115. Heart enlarged, marked visible pulsation in neck. |
| 18 | 30.5.45 | 13.3.41 | Invalided as rheumatic endocarditis. Coarctation diagnosed by medical board, 30.5.45. Working as cinema doorman. Electrocardiogram shows myocardial damage. |
| 19 | 14.8.41 | 11.4.44 | 7.5.44. Developed symptoms and signs of subacute bacterial endocarditis from which he died. |
| 20 | 27.8.45 | — | Died suddenly whilst on leave. Autopsy showed coarctation with rupture of the aorta. |

Pericardial sac distended with blood clot, ragged aperture at the back of the aorta. Heart very large, 700 g. Left ventricle much hypertrophied, muscle firm. Moderate degree of coarctation of the aorta and patchy atheroma above the aortic valves. A ragged triangular tear in the intima just above the right coronary artery. This opens into a dissecting aneurysm which has stripped off the intima over the greater part of the ascending aorta and arch. Considerable amount of blood in the coats of the vessel, and rupture has occurred into the pericardium through the back of the ascending aorta. There is nothing in any way suggestive of specific disease. Extensive atheroma of the vessels at the base of the brain.

"*Conclusions.* Coarctation of the aorta with hypertrophy of the left ventricle and a dissecting aneurysm of the first part of the aorta which has ruptured into the pericardium."

According to Blackford (1928) sudden death from rupture of the aorta during or following severe exertion is not uncommon in coarctation of the aorta. Case 3 died on the operating table from cardiac failure during operation for the coarctation. At the operation coarctation was found immediately distal to the left subclavian artery, and the intercostal vessels were much dilated. The other patient who died, Case 19, developed subacute bacterial endocarditis, after having served for 6 years. He was diagnosed as coarctation in August, 1941, but as he had no symptoms he was retained in service in a low category. Three years later subacute infective endocarditis supervened.

All the other recent war cases, as far as is known, are still living. Case 1 developed pulmonary tuberculosis with positive sputum in October, 1945, —five and a half years after discharge—but when examined for review of pension in October, 1947, was working and feeling well. Case 7, when examined in October, 1946, had developed typical symptoms of intermittent claudication, but was working as a general handyman in the building trade. Most of the remaining cases are able to carry on light work, although complaining of symptoms such as shortness of breath on exertion, pains in the chest, and giddiness.

The 1914–18 war cases which have now been followed for a long period of years will be described more fully.

*Case* 21 (Case 2 in Lewis' series) was examined for enlistment in the Royal Field Artillery in January, 1915. His occupation was described as a carman and washer in a garage and he gave his age as 35, though later documents show that his actual age on enlistment was 38 years. Physical development is recorded as good and he was passed fit for general service. He served for three and a half years including three months in France and two and a half years in Salonica. In 1916 and 1917 he was admitted to hospital on several occasions for malaria, but there is no record of any other complaints. On Nov. 26, 1917, whilst convalescing at Corfu from an attack of malaria, he was admitted to the British Military Hospital complaining of shortness of breath on exertion and slight pain in the region of the heart. The bood pressure on admission was 240 mm. Examination of the heart showed a systolic murmur at the apex transmitted to the axilla and a systolic murmur in the aortic area transmitted into the neck. The pulse was collapsing. "D.A.H." was diagnosed. On invaliding home he was transferred to hospital and in February, 1918, examination showed a soft systolic musical murmur over the tricuspid area, and a second systolic murmur in the aortic area. There was also a "soft murmur above the notch, also audible over both sides of the back from apices to level of fourth dorsal vertebra, 4 in. beyond middle line on left side and 3 in. on right side." Visible pulsation in the suprasternal notch was noted and a distinct diastolic shock over left upper front. On X-ray examination no pulsation was seen but shadow of the large vessels seemed enlarged. The Wassermann reaction was positive on Feb. 20, 1918, but negative on April 10, 1918. On June 21, 1918, he was invalided from the Service with the diagnosis of aortic aneurysm.

It is clear from the above clinical notes that many of the characteristic signs of coarctation of the aorta were present at this early date, 1917–18, as shown by the high blood pressure of 240 mm., the systolic murmur over the præcordium, also heard at the back, and visible pulsation in the neck above the sternum.

When examined by a Medical Board including a cardiologist in November, 1919, it was stated there were no signs of aneurysm. The blood pressure recorded was 250 mm. The man was next medically boarded in 1922 when the blood pressure was still 250 mm., and the heart was recorded as enlarged. The urine showed albumin and casts and the diagnosis was changed to nephritis with enlarged heart. In November, 1925, he was admitted to hospital under Sir Thomas Lewis who diagnosed coarctation of the aorta on the grounds of a collateral circulation and weakening of the femoral pulse. In September, 1939, he had an operation for removal of a renal calculus with no untoward effects. On March 2, 1940, he collapsed whilst trying to sweep the snow from the front of his house. He complained of severe pain in the left arm and back, and shortness of breath. Examination showed marked pulsation above and below both clavicles, an irregular heart due to extrasystoles and the blood pressure in the arm was 190/110 mm. and in the legs 130 mm. systolic.

In November, 1942, he was admitted to hospital with pain in the chest and hæmoptysis. He had had eight attacks of loss of consciousness during the preceding two weeks, and heart block was found. He improved with treatment and when seen by a Ministry of Pensions Medical Officer in January, 1944, he was keeping fairly

well. He died, however, in October of the same year, at the age of 68.

*Case* 22 was a greengrocer. He enlisted in June, 1915, at the age of 20, and was found fit. He served for a year and was then discharged with V.D.H. (mitral obstruction). The clinical notes recorded a presystolic thrill and murmur at the apex, increase of cardiac dullness and tumultous action of the heart. Examination a year later recorded a slight systolic murmur not conducted. On lying down various murmurs were heard. He had given up work as a stores-porter, as he found it too heavy. In 1922 a medical board reported symptoms of pain over the left side of the chest and back. The heart was enlarged clinically, the heart sounds irregular with a presystolic murmur at the apex. The diagnosis was mitral stenosis with fibrillation. When examined in 1924, complaints were giddiness, shortness of breath, and præcordial pain. The heart was moderately enlarged, rhythm normal (quinidine had been given) and there was a short diastolic murmur at the apex. In 1929 it was recorded that the man had done no work for the last year. There was some cyanosis, the heart was enlarged with systolic and diastolic murmurs at the apex, and the rhythm irregular.

In April, 1932, the man was admitted to Roehampton Hospital where coarctation of the aorta was diagnosed by the radiologist. The clinical notes recorded the apex beat an inch outside the nipple line, and a loud blowing apical systolic murmur conducted widely. No collateral circulation was detected. Marked pulsation in the vessels of the neck was present. Blood pressure in arms 178/102 mm.; femoral pulse not felt. The radiological report was "Heart considerably enlarged in all diameters; aortic arch small. Bilateral irregularity of lower margins of ribs. Appearance of coarctation of the aorta."

When seen in December, 1940, he was confined to bed with influenza. He stated he had been keeping fairly well since he had been in hospital and had been working as a shop-porter. Examination showed evidence of heart block. The pulse was regular rate 42. The blood pressure in the arms was 195/100 mm. He was last seen in September, 1941, when he was confined to bed, dyspnœic and cyanosed. Pulse rate 42. Blood pressure 180/100 mm. There was evidence of congestive failure, namely, râles at the lung bases and some œdema of the legs. This was twenty-five years after the onset of symptoms.

*Case* 23. This man enlisted in October, 1916, at the age of 24 and went to France. In May, 1918, he was admitted to hospital with the diagnosis of pyrexia of unknown origin. He complained of pains, shivering and vertigo. Examination revealed a loud systolic murmur over the præcordium, best heard at the apex and conducted to the axilla, and also heard at the back at the level of the second to fifth dorsal vertebræ. There was marked pulsations at the root of the neck. Radiological examination showed enlarged root shadows, no aneurysm, and a very greatly enlarged heart. The man was invalided with the diagnosis of V.D.H. In 1922 the man was complaining of shortness of breath, pain over the heart, and faintness at times. A systolic bruit was noted at the apex and base of the heart, but no presystolic or diastolic murmur. In 1925 his symptoms were worse. The blood pressure, recorded for the first time, was 200/90 mm. A harsh systolic murmur was noted at the apex and base.

In 1931 the man was admitted to Roehampton Hospital. It was stated he had been doing his usual work as a labourer. He complained of giddiness and faintness. The apex beat was forcible and outside the nipple line. A systolic murmur was present over the aortic and mitral areas; no diastolic murmur was present. There was forcible pulsation at the root of the neck. Blood pressure was 255/120 mm., the radiologist's report was as follows:—

> "Enlargement of the left side of the heart, usual projection of the aortic arch not shown, irregularity of the lower rib margins on both sides from the third rib downwards. Appearance that of coarctation of the aorta."

In 1935 he was only able to do light work owing to pain across the chest and fainting attacks. The blood pressure in the arm was 230/110 mm. The femoral pulse could not be felt. The Wassermann reaction was negative. The cardiogram showed left ventricular preponderance and slight widening of the QRS complexes.

In 1937 he was working as a casual labourer. There was visible pulsation under the left clavicle. No collateral vessels were detected. The brachial blood pressure was 260/145 mm. The right femoral pulse was just palpable but there was no pulsation in the left.

When last seen, in December, 1946, aged 54, he could not walk far. There was triple rhythm at the apex but no congestive failure.

These records show that patients with coarctation of the aorta may live for many years after the first appearance of signs and symptoms, and in spite of persistent hypertension. For example, Case 21 survived for 27 years after signs of coarctation and a blood pressure of 240 mm. were first recorded, and Case 23 was alive and free from congestive failure 21 years after his blood pressure had been recorded as 200/90 mm.

## Summary

A review is given of 23 cases of coarctation of the aorta, 20 of which served in the recent war, and 3 in the 1914–18 war.

These men had been fit prior to enlistment and were of good physical development. They were able to withstand the severe stress of war service for long periods before symptoms occurred.

The most constant clinical signs of coarctation were a raised blood pressure in the arms associated with a diminished femoral pulse, evidence of a collateral circulation, a systolic murmur audible over the front and back of the chest, and abnormal pulsation at the root of the neck. The

most constant radiological signs were erosion of the ribs and absence or smallness of the aortic knuckle.

Severe hypertension may persist for many years in these cases without causing heart failure and some may live a normal span of life. The later the symptoms appear the better the prognosis.

I wish to thank Dr. Prideaux, Director General of Medical Services of the Ministry of Pensions, for his permission to publish this paper.

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# THE RIGHT PRÆCORDIAL LEAD

BY

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Recent studies in clinical electrocardiography have indicated the desirability of taking multiple præcordial leads as a general routine practice. In their secondary supplementary report (1943) the Committee of the American Heart Association for the Standardization of Præcordial Leads stated that they believed that three is the least number of præcordial leads that can be regarded as satisfactory for general purposes. They suggested that those who wished to reduce the number of such leads to a minimum take leads from the C 1, C 3, and C 5 positions. All were urged to take additional leads whenever possible. Though a right præcordial lead, C1, is recommended for general use, sufficient data have not yet been obtained on this lead either in regard to the range of normal or concerning its various abnormalities. Furthermore there is no agreement as to what is the best distal electrode for general use, a matter of considerable practical importance. The Wilson central terminal electrode, the right arm, the left leg, and the right scapula has each been preferred by different investigators.

In an attempt to gain further information on the right præcordial lead C 1 and to assess its value for general use we (1) have reviewed previous reports in which such a lead was employed ; (2) have taken a series of electrocardiograms including this lead on patients without heart disease to determine the range of normal variations ; and (3) have also noted characteristic alterations in $CF_1$ in certain cardiac conditions particularly posterior wall infarction, in which the use of this lead may be helpful.

Kossman and Johnson in 1935 reported on the normal variations in multiple præcordial leads. Their subjects were 30 medical students. They used the central terminal distal electrode and the right pectoral † position. With this lead V 1 they found: no Q wave; R, 1·0 to 9·6 mm.; S, 3·4 to 24 mm.; T from −4·0 to +5·6 mm. and RS (intrinsic inflection) 6·6 to 26·8 mm. An electro-negative T wave was found in 3 out of the 30 cases in V 1.

Wood and Seltzer (1939) examined the right pectoral lead using both the right arm and the left leg as sites for distal electrodes. In regard to the QRS complex they found S dominant in 75 per cent with the use of the right arm and 95 per cent when the proximal electrode was paired with the left leg. When the right arm was used the T wave remained upright, but when the left leg was used the T wave often became inverted. Thus in $CF_1$, T was inverted in 65 per cent of normal children and 56 per cent of normal adults. With progressive enlargement of the left ventricle they found the incidence of negative T waves fell from 58 per cent to nil in grade III enlargement. In other words if there was significant enlargement of the left ventricle T was upright. Hearts displaced by a high diaphragm to give left axis deviation in the standard leads did not act in the same way. Of twelve normal pregnant women with left axis deviation in the limb leads, ten had inverted T waves and two diphasic T waves in $CF_1$. These authors concluded that in the right pectoral lead ($CF_1$) one has a means of distinguishing left ventricular enlargement from displacement of the heart due to a high diaphragm. They indicated also that dominance of the right ventricle may be better ascertained in the right pectoral lead by the use of the left leg, rather than the right arm for the distal electrode. In the former an R taller than S was more certain of this interpretation. In cases of posterior myocardial infarction they stated they derived no help from the chest leads for the limb leads required no support and the chest leads had but little to give. Deeds and Barnes (1940), Shanno (1940), and Sigler (1944), have also described characteristics of lead $CF_1$. These are summarized in the Table.

Wilson and his associates in 1944 published

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† Right præcordial and right pectoral are used synonymously for the $C_1$ position in this paper.

TABLE

RIGHT PRÆCORDIAL LEAD $CF_1$

| | Cases | P, mm | | R,* mm | | R absent | S, mm | | T, mm | | Percentage with negative T |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Minimum | Maximum | Minimum | Maximum | | Minimum | Maximum | Minimum | Maximum | |
| Deeds and Barnes, 1940 | 100 | −1·7 | +0·5 | 0·2 | 9·8 | — | 6·0 | 35·0 | −6·0 | +6·7 | — |
| Shanno, 1940 .. .. | 100 | −2·0 | +2·0 | 4·0 | 7·0 | 4·0 | 2·0 | 30·0 | −6·0 | +7·0 | 70 |
| Sigler, 1944 .. .. | 100 | −1·8 | +0·6 | 0·2 | 10·0 | — | 4·0 | 35·0 | −4·5 | +5·5 | 51 |
| Vesell and Shorr† .. | 265 | −2·8 | +0·8 | 0·2 | 7·4 | 4·9 | 2·8 | 25·0 | −4·6 | +4·2 | 69 |
| Wood and Seltzer, 1939 | — | — | — | — | — | — | — | — | — | — | 58 |

* Q was absent in all.

† RS–T was often elevated up to a maximum of 1·8 mm.

multiple præcordial lead electrocardiograms taken with the Wilson central terminal electrode in patients with myocardial infarction. The right pectoral lead V 1 was included; and they observed that with this lead in cases of high lateral, plain posterior and postero-lateral infarcts R and T of V 1 (and V 2) were unusually prominent. This was not so in postero-inferior infarction. In an article in 1946 he and his associates noted in the single case of high postero-lateral myocardial infarction that T waves became taller in leads from the right side of the præcordium.

The following is an analysis of the normal variations of the præcordial electrocardiogram taken in the right pectoral position $CF_1$. The subjects were 265 adults. There were two groups. In the first, all tracings were taken with the patient in the sitting position. This group consisted of 200 working men between the ages of 30 and 68 years, and only 11 were in the seventh decade. The majority were labourers; all were ambulant. No clinical evidence of heart disease was found in an ordinary history and physical examination and the routine four lead cardiogram (standard limb leads I, II, III, and $CF_4$) was normal by usual criteria.* In many a teleradiogram was taken and was normal. In the second group the cardiogram was taken with the patient in the recumbent position. The subjects were 65 men and women. They were patients in the hospital or office patients in private practice; their ages were from 20 to 58 years. As in the first group the heart was considered to be normal after an ordinary history, physical examination and four lead cardiogram. In the majority a cardiac fluoroscopy was done and was normal. In some the $CF_1$ lead was taken in both the recumbent and sitting position.

The cardiograph used was a string galvanometer, either the Cambridge mobile unit or Cambridge simplitrol. The electrodes were the conventional ones, 3·5 × 6 cm., of german silver. A circular electrode 3 cm. in diameter was often used for the præcordial leads and was kept in place by an elastic rubber belt. The chest electrode was placed in the fourth intercostal space at the right margin of the sternum, and the distal electrode just above the left ankle. A suitable electrode paste was employed. Standardization of the string, 1 cm. for 1 mv., was made and recorded with each lead. Measurements recorded were usually the average of several deflections or segments. Frequently a magnifying lens was used.

In view of the fact that the normal variations in $CF_1$ revealed little or no difference in the two groups, they were considered as one group of 265 cases to include males and females, and sitting and recumbent positions. P ranged from −2·8 to +0·8 mm.; 88 per cent were negative, 9 per cent diphasic, and only 3 per cent positive; the average negative P was −1·3 mm.; the average positive T was +0·5 mm. Q, interpreted to mean an initial negative QRS deflection followed by a positive, R, deflection, was not observed in any of the 265. In 13 (5 per cent) there was (see Fig. 3) an absent R wave (i.e. monophasic negative QRS wave). In several other instances where the R wave was very small, about 0·2 mm., there were occasional complexes in the same tracings in which no R was visible also forming a monophasic negative QRS (S) complex. This monophasic negative wave, S, ranged from −4·5 to −17·0 mm. with an average of −10·8 mm. R ranged in amplitude from 0·2 to 7·4 mm. The average was 2 mm. Occasionally there was some slurring of R; this was usually in small deflections and was slight. S, ranged in amplitude from −2·0 to −25·0 mm. with an average of −13·2 mm. There was infrequent slurring and this was usually with waves of low voltage and was only slight. $R^1$, was

* Criteria Committee of the New York Heart Association (1939), New York.

noted 46 times and ranged in amplitude from 1·0 to 7·5 mm. It was frequently slurred. QRS duration was the same as in $CF_4$ or in the limb leads; no noticeable difference was seen. RS–T segment was often elevated, up to 1·8 mm.; RS–T depression was not seen. T ranged from −4·6 to +4·2 mm.; it was negative in the majority of tracings, 183 of the 265, or 69 per cent: 46 were diphasic, +−; most of these were preponderantly negative. There were only 34 of the 265 (13 per cent) in which T was positive. Only 3 of the 37 females (8 per cent) had a positive T. In these three the amplitude of T was +4·0, +2·2 and +1·6 mm. with an angle alpha of +55°, +74° and +20°. In the whole series of 265 there were only 5 with T of +3 mm. or more; the average of the positive T waves was 1·8 mm.

In 15 cases in which T was negative with the patient in the sitting position a tracing was also taken, at the same time, with the patient in the recumbent position. In all 15 the T remained negative with little variation in depth. In 4 patients (2 male and 2 female) with a positive T in $CF_1$ in the recumbent position the tracing was also taken in the sitting position. In the latter position the T remained the same in two and in the other two was slightly reduced, from 1·2 to 1·0 mm. and from 1·6 to 1·2 mm.

The relationship of deviation of the electrical axis of QRS to negative T waves in $CF_1$ was examined. Axis deviation of QRS was expressed by angle alpha measured on a Dieuaide chart. In the 265 cases angle alpha ranged from −35° to +115°. No correlation of T wave negativity with deviation of the electrical axis of QRS was noted, positive and negative waves were observed almost as frequently in the range of 0° to −35° as in that of +70° to +115°.

Tracings in which a Q wave was present in limb lead III were examined; there were 123. No correlation was noted with respect to T in $CF_1$ in that both negative and positive T waves were observed in this group. There were 11 of the series of 265 in which the Q III was prominent, 25 per cent. of the largest R in the three limb leads, with no S III, and without right axis deviation. Here also both negative and positive T waves were noted in $CF_1$. In five, T in $CF_1$ was negative, in five diphasic, +−, and in one it was positive. Thus in normal subjects a negative T in $CF_1$ may occur with a prominent Q III. This is of significance in the light of the T wave findings with posterior wall infarction described later.

The effect of axis deviation on the RS deflection was the following: in 51 cases the angle alpha was between 0 and −35°. R averaged 1·4 mm. and S averaged 8·0 mm.; the relationship of R to S varied considerably in this group, from 1 to 2 to 1 to 28, with the exception of one case in which R was larger than S and R : S was as 1 to 0·4. In the group (24) with angle alpha of +80° to +115° the average for R was 1·76 mm. and for S 12·0 mm. the ratio R to S ratio ranged from 1 : 2 to 1 : 18. Thus for the whole group of 265 normals, in the axis range most to the right (clockwise), R averaged 25 per cent larger and S 50 per cent deeper than in the axis range most to the left (counter-clockwise). There was only one tracing in the whole series of 265 where R was larger than S, R = 3·2, S = −1·5 mm. and angle alpha −17°.

There was some correlation between R and S in $CF_1$ and $CF_4$. It has been pointed out that in normals R increases in amplitude as one goes from $CF_1$ to $CF_4$ or $CF_5$ and that S decreases. This was generally found to be so and R was larger in $CF_4$ than in $CF_1$ in all but 5 of the 265 cases; usually the difference in amplitude was considerable. In the 5 exceptions where R in $CF_1$ was larger it was 4·0, 4·8, 3·9, 4·8, and 4·7 mm., while R IV was 2·0, 3·0, 2·5, 3·6, and 3·0 mm. with angle alpha of +55°, +68°, +50°, +77°, +75° respectively. S was not infrequently larger in $CF_4$ than in $CF_1$.

No correlation was noted between negative T waves in $CF_1$ and amplitude of T in $CF_4$ except that T in the latter was always positive while T in $CF_1$ was negative in 69 per cent.

Fifteen cases with the cardiographic pattern of left ventricular strain were examined. The pattern of left ventricular strain consisted of a tall R I, deep S III, depressed RS–T I (often RS–T II) and frequently inversion of T I and occasionally of T II. In all 15, T was upright in $CF_1$ except for one in which the T was diphasic, −·4 and +1·0 mm. The average for T was +2·2 mm. These findings are largely in accord with those of Wood and Seltzer who found T wave negativity in $CF_1$ to be reduced in left ventricular enlargement and absent when the enlargement was marked. In the cases with the cardiographic pattern of left ventricular strain, enlargement of the left ventricle was practically always present. In this group R tended to be smaller than in normals; the average was 0·97 mm. compared with 2·0 mm. for the series of normals; in seven of the fifteen, R = O and $R^1$ was present three times. S in this group tended to be deeper than in the normal; the average was 18·4 mm. compared with 13·2 mm. for the normals.

### $CF_1$ in Posterior Wall Infarction

A white woman, 64 years of age, for several years a private patient of one of the authors, was examined at his office on April 17, 1941. Her medical condition was essential hypertension and chronic osteo-

arthritis involving the joints of the arms, legs, feet, and spine. There was no chest pain related to effort, no mid-chest pain and no undue shortness of breath. Heart sounds normal; blood pressure, 168/110; lungs clear; liver not palpable, and no œdema of the lower extremities. Fluoroscopy revealed the heart to be of normal size, shape, and position; the thoracic aorta was somewhat tortuous. A cardiogram, taken with the patient recumbent, showed some left axis deviation; angle alpha was −15°. The tracing was within normal variations. The T waves in leads I, II, and IV were upright, 3·4, 2·0, and 5·4 mm. respectively. The RS–T segment was elevated, 0·5 mm. in lead I, and depressed 0·4 mm. in lead III. In lead $CF_1$, P was −0·8 mm.; R, 0·4 to 0·8 mm.; S−6·5 mm.; RS–T, elevated 0·2 mm.; and T inverted, −0·5 to −0·8 mm. (Fig. 1). Four days later (April 21, 1941) while at home she suddenly developed severe prolonged substernal pain with the clinical picture of acute myocardial infarction. The cardiogram taken that day (Fig. 1) showed changes from the one taken four days before. RS–T in lead II became slightly elevated, with shouldering as it continued into the coved and inverted T wave. Lesser changes were present in leads I and III. $CF_1$ was not taken. Two days later, April 23, the second day of the acute attack, another cardiogram revealed progressive cardiographic changes in leads II and III characteristic of the pattern of recent posterior wall infarction. In $CF_1$, T, which 6 days before was inverted, was now on the second day of the acute attack upright and of moderate amplitude, 2·8 mm., moderately tall for $CF_1$. P remained unchanged while R became taller, 2·4 mm. compared with 0·4 to 0·8 mm. before the attack; S became slightly deeper, −8·0 mm. compared to −6·5 mm.; RS–T which was elevated 0·2 mm. prior to the attack was now depressed to 0·2 mm. below the isoelectric line. T remained upright in tracings taken ten days, 59 days and 136 days after the onset of the attack though gradually decreasing in amplitude; 2·5, 1·5, and 0·8 mm. respectively (Fig. 1). In this last tracing (136th day) T in lead II had become upright and was 0·8 to 1·0 mm. in amplitude.

Fourteen months later T in $CF_1$ again became inverted, 1·6 mm., R reduced to 0·0 to 0·2 mm., and RS–T isoelectric. The standard limb leads were similar to those taken before the attack with the exception of the presence of a small Q in lead II, 0·5 to 1·0 mm.; R I was slightly taller, R III correspondingly smaller; and T IV 2·5 mm. smaller. A cardiogram five years later (October 1947) revealed little change. A large Q wave was present in the augmented unipolar left leg lead.

Cardiograms including $CF_1$ were taken on another patient before and after an attack of acute myocardial infarction (Fig. 2). Leads II and III after the attack were characteristic of the pattern of posterior wall infarction. In lead $CF_1$, T which was negative before the attack became positive after the attack; R became taller.

These findings indicated that with acute posterior wall infarction certain changes occurred in lead $CF_1$. The changes in the T wave in this lead were striking and it was thought they might be expressive of the specific cardiac lesion and helpful in diagnosis. Accordingly, electrocardiograms in cases with the clinical picture of recent myocardial infarction and with the electrocardiogram in the limb leads characteristic of the pattern of posterior wall infarction were examined. All had a prominent Q II and Q III with inverted T II and T III; none had right axis deviation. There were 35 cases; all were patients in the hospital; 31 males and 4 females; ages ranged from 41 to 78 years. The measurements in lead $CF_1$ were: P, −0·3 to −2·0 mm.; 3 were +−; there were no positive P waves and none of unusual or abnormal configuration. Q waves were not present. R, 0·5 to 9·0 mm.; no absent R; the average was 3·9 mm. and taller than in the series of normals where the average was 2·0 mm. S, 0·0 to −19·2 mm.; average −8·2 mm. smaller than in the normal series where the average was −13·2 mm. $R^1$ was present twice; each 2·0 mm. QRS duration appeared unaltered. RS–T was depressed in 6; 0·2, 0·8, 1·0, 1·0, 1·5 and 1·5 mm. below the isoelectric base line; in two, J was the lowest point of the segment and the ascending limb of the T wave gradually rose from it. In nine, RS–T was elevated 0·2 to 1·8 mm. T was positive in all 35 cases and the range was from 1·0 to 9·0 mm. with an average of 3·1 mm. T was generally taller than in normals where the average positive T was 1·8 mm. and in only 5 of the 265 normals was T 3 mm. or taller. Negative T waves were not observed. The configuration and duration of the T did not appear abnormal.

Observations were not made on the duration of these changes in $CF_1$ in the 35 patients after their discharge from the hospital. However, in the first case described above the T wave changes were noted to last at least 4 months. In a cardiogram 14 months after the attack the T wave was again negative.

Thus it appears that lead $CF_1$ reflects changes in the posterior wall of the heart, exhibiting characteristic alterations with infarction of this area. The cardiographic changes are of opposite sense to those observed in the unipolar left leg lead in this condition, and may be contributed at least in part by the left leg component. R became taller and S

deeper. RS–T often was depressed below the base line. This did not occur in normals. The most striking alteration was in the T wave which was positive in all such cases though in the normal T was positive in only 12 per cent and even less in females. This T wave alteration in $CF_1$ could be

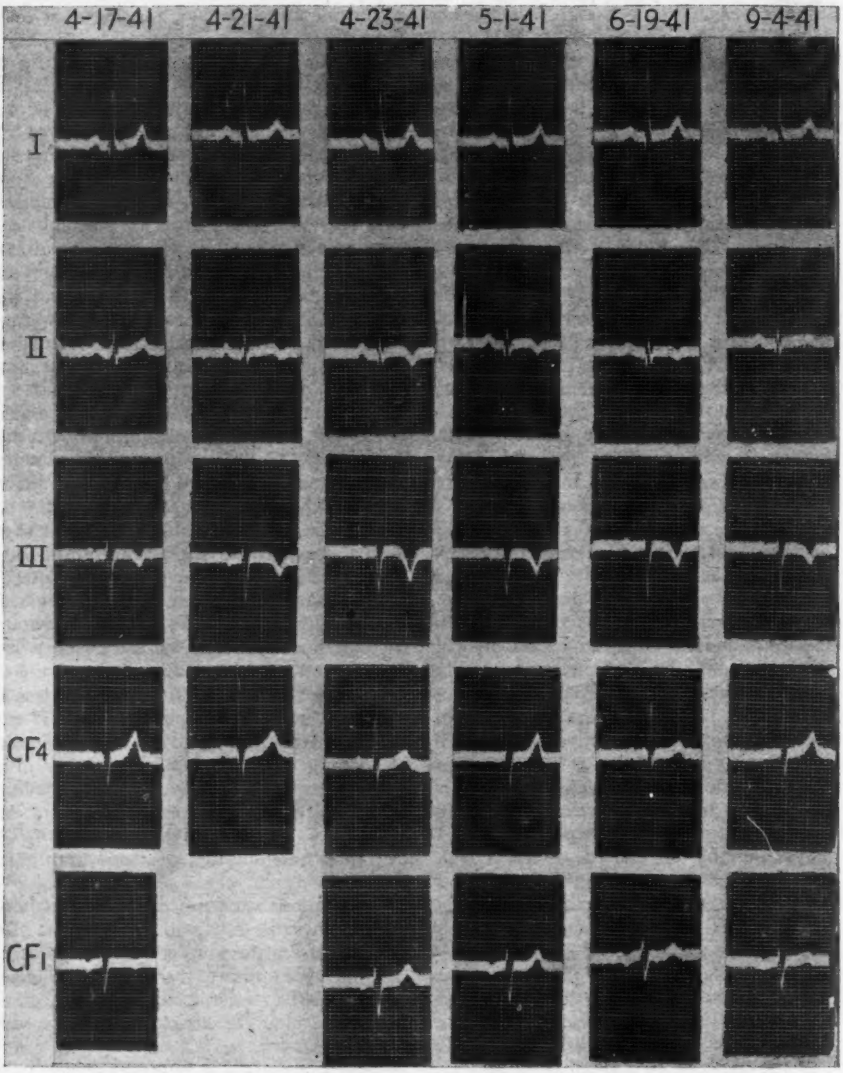


FIG. 1.—Cardiograms of a patient who had acute myocardial infarction on April 21, 1941. Serial changes in the limb leads II and III of subsequent tracings conformed to the cardiographic pattern of posterior wall infarction. The cardiogram on the day of the attack, April 21, 1941, did not include $CF_1$. In $CF_1$, four days before the attack T was inverted, R small; two days after the attack, April 23, 1941, T was upright 2·8 mm.; gradually becoming smaller in the tracings on the tenth, fifty-ninth, and one hundred and thirty-sixth day after the attack. R was taller after the attack.

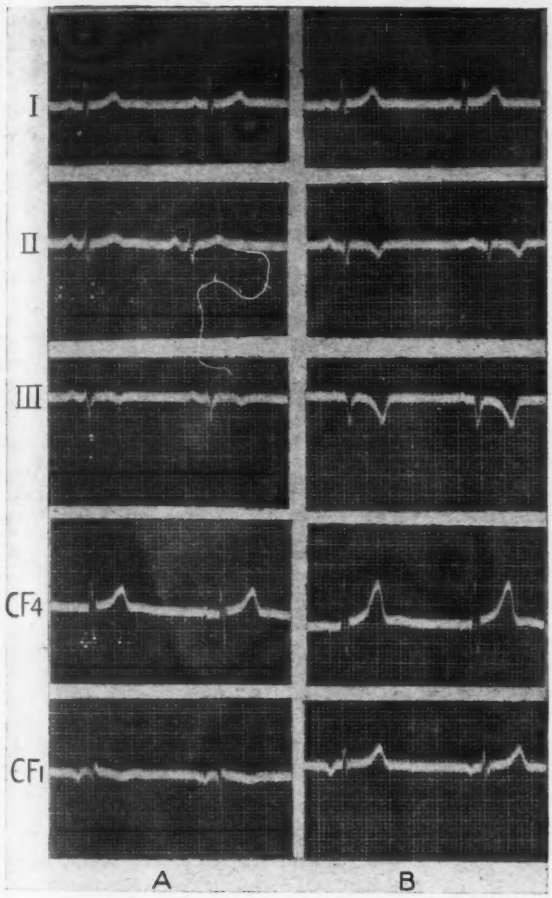


FIG. 2.—Cardiograms (A) before and (B) after attack of acute myocardial infarction. Serial changes in limb lead II and III and $CF_4$ conform to the cardiographic pattern of posterior wall infarction. In A lead $CF_1$, R=O and T is inverted; in B lead $CF_1$, R is upright, 3·8 mm., and T is upright, 4 mm.

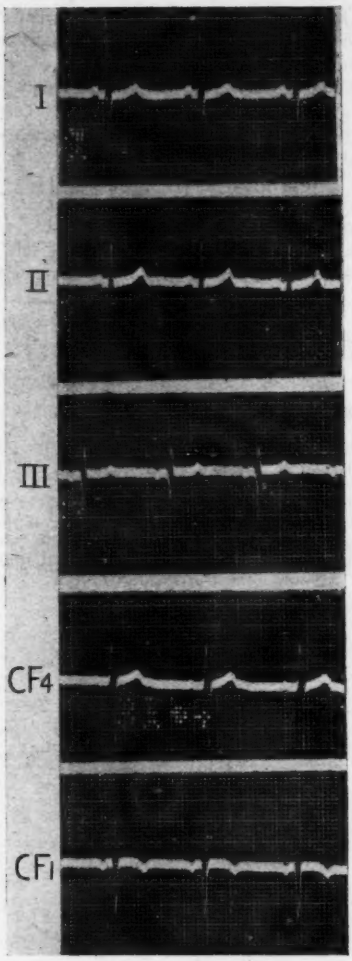


FIG. 3.—Cardiogram showing absent R in $CF_1$; also inversion of T. In 15 cases in the series without heart disease R=O in $CF_1$, frequently with inversion of the T in this lead.

of use in the diagnosis of recent posterior myocardial infarction. It may be particularly helpful where changes in the limb leads especially lead II, are equivocal or absent and also in the evaluation of Q III. A negative T wave in $CF_1$, which occurs in about 70 per cent of normal adult electrocardiograms, would, from the above, be important evidence against the presence of recent infarction of the posterior wall of the heart.

## SUMMARY AND CONCLUSION

Data on the right præcordial lead $CF_1$ have been reviewed. The characteristics of $CF_1$ in a group of 265 patients without heart disease have been described.

Variations of the T wave, in particular, in $CF_1$ are described in relation to changes in position of the patient, electrical axis of QRS, presence of a prominent Q III, and in left ventricular strain.

In two patients, $CF_1$ was taken before and after the occurrence of a posterior myocardial infarction and revealed the development of characteristic changes. These characteristic changes in $CF_1$ were found in each one of a series of 35 cases of recent myocardial infarction of the posterior wall. The use of lead $CF_1$ in the diagnosis of posterior wall infarction is discussed.

Usefulness of $CF_1$ has been indicated in (1) locating the side of bundle branch block, with $CF_4$ or $CF_5$ (Wilson); (2) evaluation of left ventricular enlargement, particularly from displacement by the diaphragm; (3) possibly of right ventricular enlargement (Wood and Seltzer), and (4) in posterior wall infarction. It has also been used for better recording of auricular activity and by Ellis and Brown (1946) as an aid in the diagnosis of tricuspid insufficiency.

The information obtained from a right præcordial lead C 1, and in particular $CF_1$, described above, indicate it to be of sufficient value to recommend its regular use with other præcordial leads in routine electrocardiography for general purposes.

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# CORONARY ARTERY ANEURYSM WITH OCCLUSION DUE TO A CALCIFIED THROMBUS

BY

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## Case Report

*Clinical History.* In January 1947 a Cypriot officer, 23 years of age, fell unconscious whilst playing football, and was dead when examined by a doctor five minutes later. His unit medical officer had known him for five years and stated that, apart from appendicectomy in 1945 and bacillary dysentery and folliculitis of the face and neck in 1946, he had been well and had led a normal active life. He had had further trouble with his folliculitis for two weeks before his death and was attending daily for local treatment, but he did not complain in any way about his general health and played football regularly.

*Post-mortem examination.* A well encapsulated loculated hydatid cyst, 4 cm. in diameter, was situated in the upper part of the right lobe of the liver and the peritoneum covering the cyst was thickened. Apart from the findings in the cardiovascular system the other viscera were normal.

The left coronary artery, 5 mm. from the aortic orifice, contained a pear shaped mass, 1·8 cm. long and varying in width from 0·3 to 1·0 cm., which was calcified peripherally to form a cyst-like structure and centrally it consisted of organizing laminated blood clot. In this region the artery was dilated and the walls thinned forming a cavity or recess in which the cyst lay with its broader distal end impacted in the narrow lumen beyond. There was very little atheroma and the vessel walls showed a few scattered plaques only (Fig. 1).

The right coronary artery, about 1 cm. from its origin, was also dilated locally and there was a small indentation or pocket in the wall where marked atheromatous changes had developed. The vessel was patent. The myocardium showed softening in the posterior part of the wall of the left ventricle but there was no evidence of old infarction. All the valves were normal. Unfortunately the specimen was accidentally destroyed before histological preparations could be made. There can be no doubt that sudden impaction of the calcified cyst had caused occlusion of the artery and led to death.

## Discussion

It was at first thought that the calcified cyst might be related to the hydatid cyst of the liver, but a

Fig. 1.—Diagrammatic drawing of the anterior aspect of the heart. The pulmonary artery has been removed and the aorta pulled forward to demonstrate the whole length of the left coronary artery. Within 5 mm. of the ostium there is an aneurysmal dilatation containing a pear-shaped calcified cystic mass.

hydatid seedling would be unlikely to lodge in a large artery; it would be swept on into the smaller capillaries supplying the myocardium. A hydatid cyst of the myocardium is rare; a hydatid cyst within a coronary vessel would seem to be an impossibility. The germinal epithelium would be found within the cyst but it contained old blood clot only. Furthermore the condition of the right coronary artery made it clear that the case was one of bilateral aneurysm. Within the left aneurysm a thrombus must have developed and subsequently became partially calcified. Monoher (1938) reported a case in which an oval coronary aneurysm arising 3 mm. beyond the aortic opening contained laminated clot.

Aneurysms of the coronary vessels are well known rarities but we have been unable to discover any other reported instance of sudden death following coronary artery occlusion by a foreign body lying within the lumen of the vessel. The first case of coronary aneurysm was reported by Bougon in 1812, and Packard and Wechsler (1929) collected 29 reported cases and added one of their own. From an analysis of these cases they concluded that the condition is three times as common in men as in women and that in the vast majority of cases the aneurysm is single and is usually situated on the left coronary artery within 2·5 cm. of its ostium. Sudden death is common and practically always due to rupture of the aneurysm; gradual heart failure is the cause of death in the others. They found no typical symptoms referable to the aneurysm itself, indeed in many cases no complaints of illness were known at the time of their sudden death. In a consideration of the ætiology they classified the cases into a mycotic-embolic group, occurring in association with acute or subacute endocarditis of the aortic valve and into an arteriosclerotic group where a marked coronary sclerosis was present. Aneurysms due to periarteritis nodosa were carefully excluded from the review. Syphilitic mesaortitis was responsible for three cases in the series. In the second group they believe that the situation of the aneurysms, immediately beyond the orifice, suggests that arteriosclerosis probably precedes the aneurysmal dilatation; but when one considers the frequency of coronary sclerosis and the rarity of aneurysms, such an ætiology, even with associated hypertension, is difficult to accept. Harris (1937) in reporting a case of circoid aneurysm of the right coronary artery advanced a deficiency in the elastic lamella as a probable cause of the dilatation. More recently Rigdon and Vandergriff (1943), reviewing reported cases, consider the possible ætiology in young persons may be a solitary arteriosclerosis of the coronary vessels, though they favour congenital anomalies as more probable. They instance the work of Forbus (1930) who studied the aneurysms that develop at the point of bifurcation of the medium sized cerebral arteries, and demonstrated defects in the vessel walls; he found similar deficiencies in coronary vessels. Rigdon and Vandergriff conclude that coronary aneurysms develop in association with defects in the muscular coats located at either the point of bifurcation or at a point where a branch leaves the parent vessel.

In our case there was no evidence of syphilis, mycotic-emboli, or periarteritis nodosa, and it was clear from the advanced calcification of the thrombus that it was older than the early atheroma and it is considered that the latter was rather a resultant than a causative factor. In the absence of histological proof the exact ætiology must remain a matter for speculation but we are of the opinion that the bilateral aneurysms were probably due to congenital defects in the vessel walls.

## Summary

A case of bilateral coronary aneurysm in a man aged 23 is reported. Sudden death was due to impaction of a calcified thrombus in the left coronary aneurysm. The ætiology is discussed, and reasons are given for regarding it as congenital.

We wish to thank Major General F. Harris, Director of Medical Services, Middle East Land Forces, for permission to publish this case and Brigadier C. G. Parsons, Consultant in Medicine, for his help and advice.

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# HEART BLOCK IN OSTEITIS DEFORMANS

BY

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The simian figure of the country gentleman from the north of England, who was Sir James Paget's first reported case (1877) of osteitis deformans, hobbles still through all accounts of this disease: he exemplifies most of the known complications. Paget himself learnt little more from the twenty-two other cases he saw (1882, 1889), and since then a forest of at least 700 articles, covering over 2000 cases, has added little wholly new to the original description.

The basic problem of the ætiology still eludes us. Schmorl (1926, 1930, 1932) has shown the disease to be less rare than Paget thought, and with many others (Higbee and Ellis, 1911; Knaggs, 1926; and Jaffe, 1933, the chief) has extended our knowledge of the morbid anatomy. Da Costa *et al.* (1915) questioned, and Koller (1946) seems to have refuted, Paget's denial of all hereditary element. Packard *et al.* (1901) and Knaggs (1926) questioned, but Coley and Sharpe (1931) established beyond question, Paget's claim of a relationship to bone sarcoma. Of perhaps more fundamental importance are the demonstrations, by methods not available to Paget, of the high blood phosphatase by Kay (1930) and of the extraordinarily high blood flow through affected bones by Edholm, Howarth, and McMichael (1945).

The interest in the vascular relations of the disease following the latter work encourages us to report two cases of a complication that has so far been overlooked. In both cases calcification of the interventricular septum, consequent we believe upon the Paget's disease, interrupted the bundle of His and produced heart block. This seems to be no coincidence: a recent series (Windholz and Grayson, 1947) of 12 cases of heart block (in all of which septal calcification was demonstrated radiologically) included 2 cases of Paget's disease.

## Case Notes

*Case* 1. An unmarried woman of 71 who worked as a chambermaid was admitted under the care of Dr. Paul Wood in January 1936. She stated that since youth she had suffered from pains in her legs. In 1919, at the age of 54, this pain became much worse and her legs became bowed, and at the same time her left clavicle enlarged. She was able to get about with difficulty but continued working for two years, after which she was only able to move with crutches. The disease progressed and when admitted in 1936 she was quite unable to get about. *On examination* she showed the typical picture of Paget's disease with bowing and thickening of both femora and tibiæ and the right ulna and clavicle. There was a hard mass in the connective tissues just lateral to the crest of the left ilium. The skull and spine were not deformed. Skiagrams confirmed the diagnosis of Paget's disease and showed the subcutaneous mass to be calcarious. The blood count was within normal limits, blood calcium was 9·2 mg. per 100 ml., blood phosphorus 9·1 mg. per 100 ml., and blood phosphatase was 52 King-Armstrong units but later fluctuated between 60 and 70 units. The pulse was 80, the B.P. 175/85. The apex beat was forceful four and a half inches from the midline. There was a rough systolic murmur at the apex and a soft systolic murmur at the aortic area. She was transferred to the chronic block and about 18 months later began to have attacks of unconsciousness preceded by a sensation of fluttering in the abdomen. A cardiogram during these attacks showed complete heart block (Fig. 1) with a ventricular rate of 30 and an auricular rate of 120. Between attacks there was right bundle branch block. These Stokes-Adams attacks continued intermittently until December 1937 when she died in one.

*Autopsy* was carried out 39 hours after death. Advanced Paget's disease was seen in the right femur, tibia, clavicle, pelvis, and sternum. There was Paget's disease without significant deformity in the spine. There was no apparent disease in the ribs (Fig. 2), right fibula, skull, or left clavicle. Other bones were not excised. In the right clavicle

the disease had progressed to almost ivory-like sclerosis but in all the other affected bones osteoporosis was in advance of sclerosis and the bones were soft and extremely vascular.

The mass in the subcutaneous tissues proved to be a focus of amorphous calcification 8×6×3 cm. The heart weighed 585 g. and showed muscular hypertrophy. The coronary arteries were injected

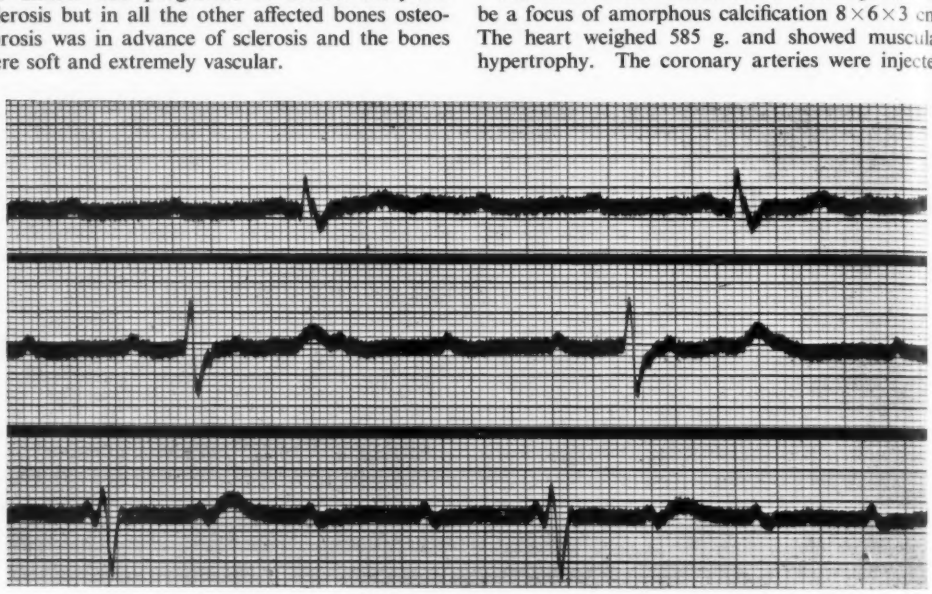


FIG. 1.—Case 1. Electrocardiogram during a Stokes-Adams attack.

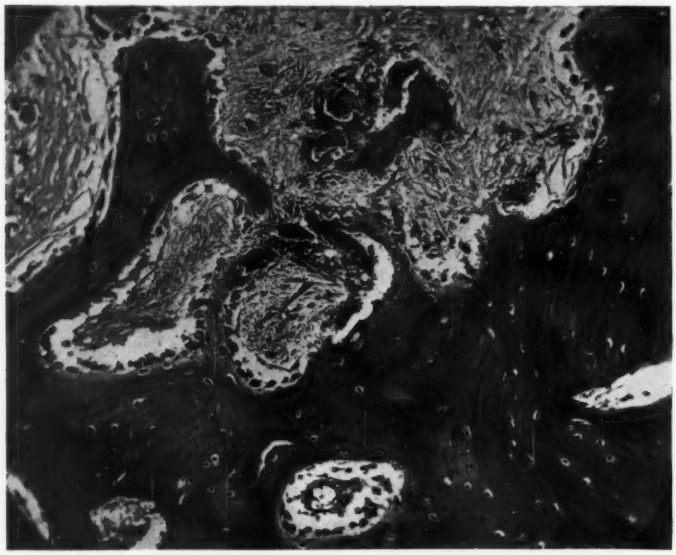


FIG. 2.—Case 1. Rib, showing osteoblastic and osteoclastic activity, vascular fibrous marrow, and irregular cement lines. Stained with H. and E. Magnification: ×112.

with barium gelatine mixture and skiagrams showed normal lumina. At the base of the mitral valve, in the connective tissues at its attachment, there was a bar of calcification extending the length of the posterior cusp and spreading on to the base of the interventricular septum to involve the pars membranacea septi (Fig. 3). Apart from this the mitral leaflets were healthy as were those of the other valves.

*Microscopic section* confirmed the gross findings. The calcification in the heart was amorphous and was surrounded by fibrosis. There was Mönckeberg sclerosis in the iliac artery. The kidneys did not show hypertensive changes and the degree of cardiac hypertrophy (585 g.) remained unexplained. In the light of Case 2, one is tempted to suggest that this too may have been an example of high output hypertrophy due to a vascular shunt through bones.

*Case* 2. An old-age pensioner, aged 74, was admitted in October 1947 under Professor

FIG. 3.—Case 1. X-ray of the septum after barium gelatine injection of vessels, showing the intact blood supply and calcification involving the pars membranacea.

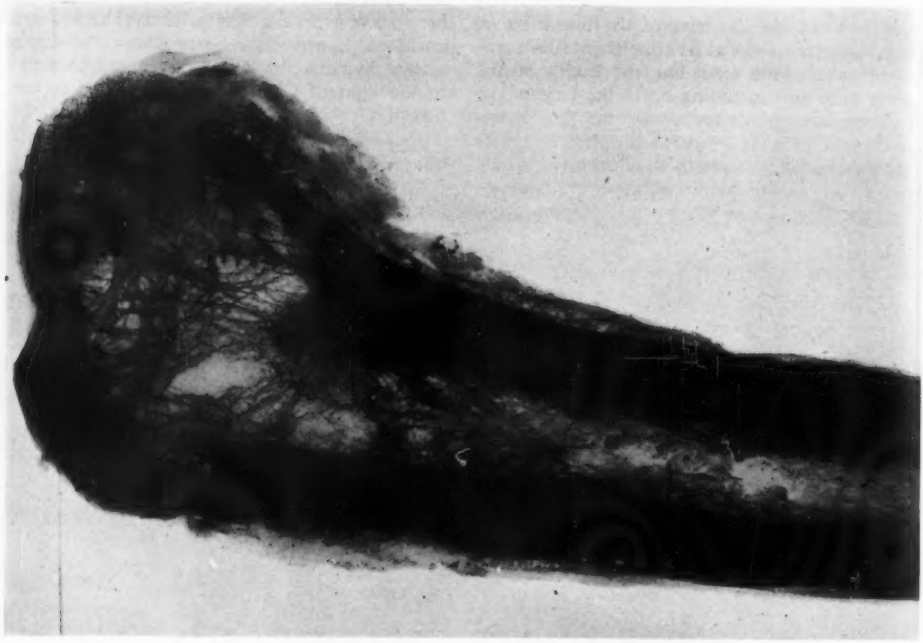


FIG. 4.—Case 2. X-ray of lower end of femur (post-mortem specimen) showing advanced Paget's disease.

FIG. 5.—Case 2. Electrocardiogram showing complete heart block with ectopic beats.

J. McMichael. A known case of Paget's disease for many years he had been bedridden for 2 to 3 years. There had been dyspnœa, orthopnœa, and mental changes for three weeks, and œdema for one week. *On examination*, there was orthopnœa, widespread œdema, an enlarged tender liver, fluid at the right base, and jugular venous pressure 10 cm. above the sternal angle. The skeleton showed gross deformity of the skull, limbs, and clavicle: radiographs confirmed extensive Paget's disease (Fig. 4) and showed also extensive arterial calcification. Pulse rate, 34; B.P. 120/60. Apex beat four and a half inches to left of midline. A blowing systolic murmur over whole præcordium. A cardiogram (Fig. 5) showed complete block with coupled ectopic beats (Dr. Paul Wood). Alkaline phosphatase 132 units (King-Armstrong). Blood urea 54 mg., bilirubin 1·5 mg. each per 100 ml. Cardiac catheterization was only partially successful, but an abnormally high oxygen content of the venous blood, especially in the jugular vein, was demonstrated. Treatment of the cardiac failure proved ineffective and he died on Nov. 3, 1947, twelve days after admission.

*Autopsy* was carried out 11 hours after death. The skeletal changes of Paget's disease were gross and typical, involving the cranium, all bones of the trunk, and all long bones except the fibulæ.

*Microscopically* the bone changes were typical and indicated widespread activity (Fig. 6). Vascularity was obvious both macro- and microscopically. The heart weighed 435 g.: there was moderate hypertrophy of both left (1·8 cm.) and right (0·7 cm.) ventricles. The posterior mitral cusp was contracted into a solid calcified ball: all cusps of the aortic valve, with both the aortic and the mitral rings, were thickened and patchily calcified: the process had extended into the upper posterior two-thirds of the membraneous septum. The extent of the lesions is best seen in an X-ray of the specimen (Fig. 7). The anterior descending coronary branch was blocked near its origin and the block calcified, but there was no infarct. Histological examination of the heart added little to the naked eye findings: in the region of the calcification the bundle of His was quite unrecognizable, though Purkinje fibres in the muscular septum appeared healthy. There was extensive medial calcification with relatively mild atheroma in arteries everywhere (Fig. 8). Findings in other systems included cystitis and an old pyelonephritic atrophy of the right kidney (55 g.), calcification of the right cupola of the diaphragm, and an old collapse of the lower lobe of the right lung. The thyroid, pituitary, and adrenals were normal. The parathyroids were of average size but showed an almost complete absence of oxyphile cells.

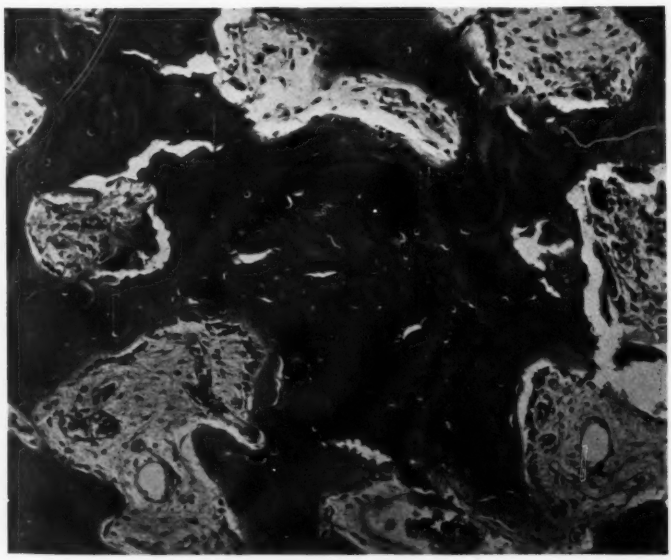


FIG. 6.—Case 2. Rib, showing active Paget's disease. Stained with H. and E. Magnification: ×112.

## DISCUSSION

Calcification of the heart valves and rings, though often described, has not been shown to be significantly commoner in Paget's disease than in controls of the same age. We have, therefore, collected some data on this point.

Our departmental records contain 13 cases of generalized Paget's disease. Six of their hearts showed valvular calcification (see Table I). To this we have added 30 reported cases. Adequately detailed accounts are rare: most articles are purely clinical (including nearly all the larger series—Gutman and Kasabach, 1936; Newman, 1946; Kay *et al.*, 1934; Sugarbaker, 1940; O'Reilly and Race, 1932; Looser, 1926; and others (especially Schmorl) deal only with the bones. A search of 170 of the readily available articles,

FIG. 7.—Case 2. X-ray of a transverse slice of the heart containing the valves, showing irregular calcification involving especially the posterior part of the mitral ring and extending on to the membranous septum.

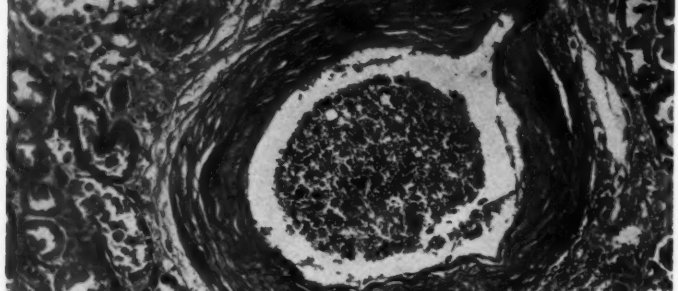


FIG. 8.—Case 2. Left kidney, showing medial calcification in a small artery. Stained with H. and E. Magnification: ×112.

covering some 820 cases, has yielded only 30 autopsied cases in which the disease was generalized, the diagnosis clear, and the heart either described or reasonably to be assumed normal; several of these showed calcification (Table II). Combining our 13 with the 30 reported cases of Paget's disease we find that 17 out of the 43 (39 per cent) showed valvular calcification. In a control series of 223 autopsies of similar age distribution from our departmental records 17 showed valvular calcification (8 per cent). A more detailed analysis of these figures is shown in Fig. 9, which also gives the figures for non-calcarious valvular sclerosis.

The two tables give details of the cases from which Fig. 9A was constructed. The eleven cases in Table I make, with the 2 already described, a total of 13 seen in twelve years at this hospital among 4300 autopsies (0·3 per cent). No special search for the disease had been made during this time and all the cases listed were extensive and unmistakeable: cases with small and doubtful lesions have not been included. It may be mentioned in passing that the absence of oxyphile cells in otherwise normal parathyroids, noted by Davie and Cooke (1937), has been confirmed in four cases of five examined.

In all those cases in Table II where calcification of valves has not been specifically noted it has been assumed absent: but in several of the older cases (e.g. Goodhart's and Silcock's) the wording leaves some room for doubt.

Two conclusions can be drawn from this comparison. Cardiac calcification is about five times as common in Paget's disease as in controls, and the total incidence of valve disease is about the same (the difference between the two sets of figures is statistically significant but in view of the mixed nature of our data this is perhaps not to be relied on). The implication of this is that whilst collagenous sclerosis of the heart valves is fairly common in old people, those with Paget's disease have an exaggerated tendency to calcify. Necrotic tissue or hyaline collagen have a natural tendency to calcify possibly because of their low $CO_2$ tension but in Paget's disease there is evidence of an additional factor in the form of an unduly labile calcium metabolism. Wells and Holley (1942) recorded a case of Paget's disease in which heavy doses of Vitamin D produced fulminating metastatic calcification, and Seligman and Nathanson (1945) recorded a similar but slower result following relatively small doses. Furthermore, Reifenstein and Albright (1944) recorded two cases of Paget's disease in which immobilization by fracture combined with an ordinary hospital milk diet produced a dangerous elevation of blood calcium. This last observation

TABLE I

PERSONAL CASES OF OSTEITIS DEFORMANS

| No. | Sex and Age | Bone changes | Cause of death | Weight of heart (g.) | Valve lesions | Arterial calcification noted |
|---|---|---|---|---|---|---|
| 3 | F, 56 | Generalized | Carc. rectum | Normal | — | — |
| 4 | F, 60 | Generalized | Renal calculi | 235 | — | — |
| 5 | M, 65 | Pelvis only | Fracture of spine | 340 | — | — |
| 6 | M, 67 | Generalized | Staphylococcal endocarditis | 600 | Fibrous thickening of posterior cusp of mitral valve, with large recent vegetations | Aorta |
| 7 | M, 68 | Generalized | Paraplegia (due to Paget's disease of vertebræ) | 400 | One aortic cusp calcified | — |
| 8 | M, 70 | Generalized | Multicentric tumour and high output heart failure | 475 | Calcified aortic ring | Aorta |
| 9 | F, 70 | Generalized | Meningioma | 300 | — | Aorta |
| 10 | F, 71 | Long bones of legs only | Carc. bladder | 185 | (Brown atrophy) | — |
| 11 | M, 75 | Generalized | Carc. rectum | 335 | Calcified aortic ring | Posterior tibials |
| 12 | M, 83 | Skull and vertebra only | Coronary thrombosis: terminal femoral artery thrombosis with gangrene | 410 | — | Aorta |
| 13 | M, 86 | Generalized | Urethral stricture | 350 | Aortic and mitral rings and aortic cusps calcified | Aorta |

TABLE II
REPORTED CASES OF OSTEITIS DEFORMANS

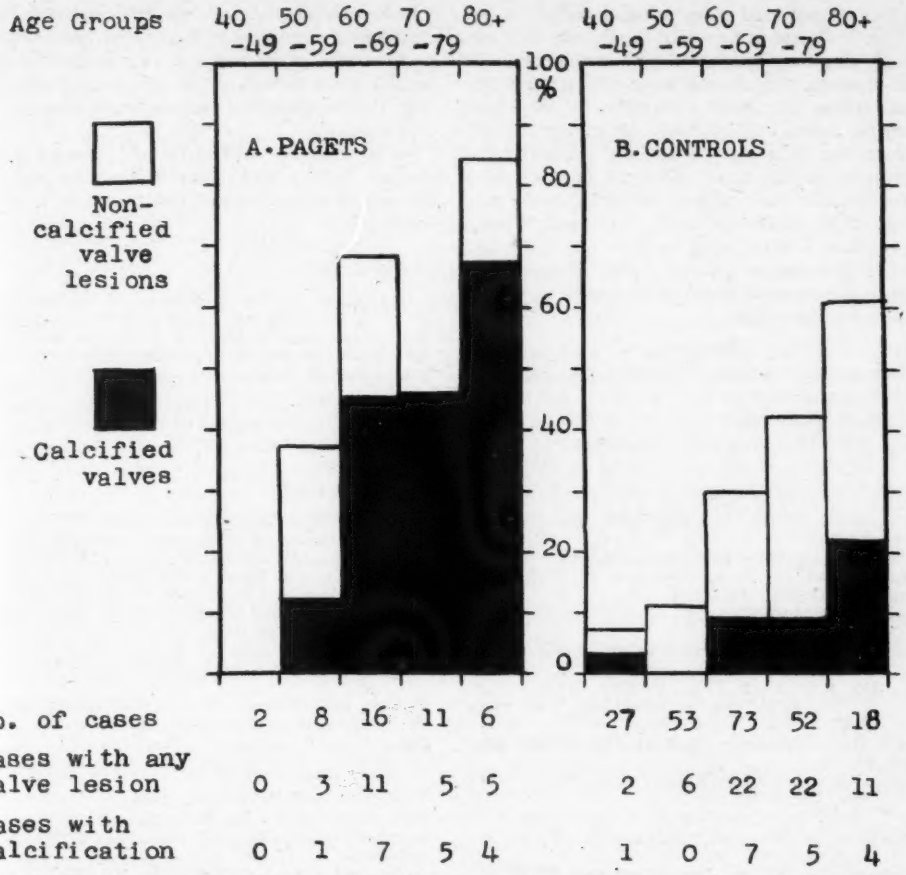
| Author | Sex and Age | Cause of death | Valve lesions |
|---|---|---|---|
| Davie and Cooke, 1937 (II) | M, 42 | Multiple sarcomata | — |
| Gilles *et al.*, 1894 .. .. | M, 49 | Unexplained heart failure | — |
| von Albertini, 1928 .. .. | M, 51 | Sarcoma of femur | — |
| Askanazy, 1904 .. .. | F, 51 | Multiple fractures | " Whitish thickening of single valve cusps " |
| Robinson, 1886 .. .. | M, 52 | Cerebellar tumour | — |
| Bartlett, 1910 .. .. | F, 53 | Broncho-pneumonia | Slight thickening of mitral cusps |
| Gruner, *et al.*, 1912 .. .. | M, 56 | Sarcoma of humerus | — |
| Ely, 1923 .. .. .. | M, 57 | Gallstones and cholangitis | — |
| Wells and Holley, 1942 .. | M, 59 | Metastatic calcification | Calcification of aortic and mitral valves, septum and atrial endocardium |
| Wilks, 1869 .. .. .. | M, 60 | Cor pulmonale and pericarditis | — |
| Goodhart, 1877 .. .. | F, 60 | Multiple sarcomata | " Pulmonary valves were thick and aortic valves had a few vegetations on them " |
| Packard *et al.*, 1901 .. | M, 62 | Sarcoma of skull | — |
| Levi, 1897 .. .. .. | F, 62 | Heart failure | Thickened mitral and tricuspid valves, calcified aortic valve |
| Higbee and Ellis, 1911 .. | M, 63 | Heart failure | Calcified aortic and mitral valve and septum |
| Robin, 1894 .. .. .. | M, 64 | Heart failure | " Mitral insufficiency " |
| Clegg, 1937 .. .. .. | M, 64 | Heart failure | Calcification of aortic and mitral valves |
| Davie and Cooke, 1937 (I) | F, 64 | Sarcoma of tibia | — |
| Cayley, 1877 .. .. .. | M, 65 | Carcinoma of lung | Calcification of aortic and mitral valves |
| Jamieson, 1897 (II) .. .. | M, 65 | Heart failure | Calcification of aortic cusps |
| Paget, 1877 (I) .. .. | M, 68 | Sarcoma of radius | Calcification of mitral valve |
| Lunn, 1887 .. .. .. | M, 68 | Bulbar palsy | Thickening of mitral valve |
| Stilling, 1890 (II) .. .. | F, 70 | ? | — |
| Miller, 1943 .. .. .. | F, 73 | Meningioma | — |
| Jamieson, 1897 (I) .. .. | F, 73 | Gangrenous leg ulcer | Calcification of aortic cusps |
| Hudelo and Heitz, 1901 .. | M, 75 | Pericarditis | — |
| Stilling, 1890 (I) .. .. | M, 77 | Emphysema and cor pulmonale | — |
| Smith, 1928 (I) .. .. | F, 81 | Heart failure following fracture of hip | Calcification of aortic and mitral valves |
| Silcock, 1884 .. .. | F, 84 | Strangulated hernia | " Chronic degenerative changes " |
| Cone, 1922 .. .. .. | M, 85 | Papilloma of bladder with stone | Calcification of aortic and mitral valves |
| Stilling, 1890 (III) .. .. | F, 92 | ? | Calcification of aortic cusps |

suggests that when Paget's disease is sufficiently crippling to immobilize the patient the risk of metastatic calcification is enhanced—a factor that was present in both of our cases. It seems to us probable that the presence of calcification in the fibrous tissue at the base of the valves may well excite further fibrosis in the neighbourhood and thus become a progressive lesion. This would explain the extension of the process to the membranous septum and justify us in regarding the heart block as a true complication of Paget's disease and not a mere coincidental calcification in a patient with heart block due (a well recognized occurrence) to simple fibrosis. We hope that by drawing attention to these cases we may stimulate a further study that will confirm or refute this.

### THE VASCULAR SYSTEM IN PAGET'S DISEASE

Vascular disease is extremely frequent in Paget's disease. Any collection of case histories makes this obvious—Kay *et al.*'s especially well. The vascular lesions have been supposed responsible for the bone disease: Léri (1926) believes the lesions of the nutrient arteries to be responsible, Cone (1933) regards the lesion as a sort of chronic venous congestion of bone. But many of the younger cases show Paget's disease without any vascular lesion (e.g. our Cases 3 to 5 in Table I) and clinically it has often been clearly demonstrated that the heart lesion develops long after the bone lesion (Paget's first case, for instance, also those of Haguenau *et al.*, 1934, and of Thibierge 1893).

The assaults of Paget's disease on the heart and




| | A. Paget's | | | | | B. Controls | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| No. of cases | 2 | 8 | 16 | 11 | 6 | 27 | 53 | 73 | 52 | 18 |
| Cases with any valve lesion | 0 | 3 | 11 | 5 | 5 | 2 | 6 | 22 | 22 | 11 |
| Cases with calcification | 0 | 1 | 7 | 5 | 4 | 1 | 0 | 7 | 5 | 4 |

FIG. 9.—The histograms show the percentage incidence by age groups of calcarious and non-calcarious valve lesions in 43 cases of Paget's disease and 223 controls. The actual number of cases is given below.

vessels are various: indeed it is surprising that so many patients reach extreme old age. We believe the principal damaging factors to be as follows.

(a) *High cardiac output* caused by the shunt through the very vascular bones, implicit in the observations of Redman (1889) on the pulse and of Klippel and Weil (1911) on localized hyperthermia over affected bones, but first clearly demonstrated by Edholm *et al.* (1945).

(b) *Arterial calcification*, as first demonstrated radiologically by Béclère in 1901. O'Reilly and Race (1932) found it in 43 per cent of their cases and although they give no control figures they were satisfied that this represents a significant increase above what might be expected in patients of the same age. The finding has been commonly called "arteriosclerosis" and might be supposed to consist of either Mönckeberg's sclerosis or calcarious atheroma. Since, however, occlusive vascular disease is certainly not unduly frequent in Paget's disease (there is only one certain arteriosclerotic gangrene in our 820 collected cases and no coronary death in our 43 autopsies) it seems reasonable to assume that the increased incidence of radiological shadows is due to Mönckeberg's sclerosis.

(c) *Thoracic deformity* producing cor pulmonale. This is surprisingly rare, though one of the earliest cases (Wilks, 1869—which is also Paget's Case 4 of 1877) died in this way, and perhaps also Stilling's Case 1 (1890).

(d) *Valve calcifications* discussed in this paper.

(e) *Heart block*. This is perhaps not so rare as

the lack of reported cases would indicate. Cases of Paget's disease in time join the chronic sick and, though they may be studied well at some stage of their progress, they do not usually die under supervision unless they have a sarcoma or some such dramatic lesion. Heart block in a heart already embarrassed by a high output and calcified valves is probably rapidly fatal: there are quite a number of unexplained heart failures among published cases (Clegg, 1937; Higbee and Ellis, 1911; and S. Paget, 1884) which it is tempting to think may have been fatal Stokes-Adams attacks. With increasing interest in the causes of death in old people more cases may well come to light.

SUMMARY

Two cases of osteitis deformans are reported in which calcification of the membranous interventricular septum caused complete heart block.

A survey of published papers has revealed only two similar cases but there are reasons for suggesting that this complication may be more frequent than this indicates.

In 30 published and 11 further personal cases of Paget's disease, calcification in the heart was found to be significantly commoner than in a control series.

Our thanks are due to Professor J. McMichael and Dr. Paul Wood for much help with the clinical aspects of these cases, and to Mr. J. R. Baker and Mr. J. Griffin for the sections and Mr. E. V. Willmott for the photographs used in illustration.

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# VENTRICULAR COMPLEXES IN HEART BLOCK

BY

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In electrocardiograms of complete A-V block, the ventricular complexes may be of normal supraventricular type, of typical bundle branch block (B.B. block) type, or may exhibit lesser degrees of widening and notching of QRS and T wave changes. The association of B.B. block with complete A-V block is recognized as common. Campbell (1944) found B.B. block in 19 of 64 cases of complete block. Similar findings have been recorded by White and Viko (1923) who found evidence of intraventricular block in 15 out of 27 cases of complete heart block, and by Fatzer (1946) who described intraventricular block in 60 per cent of cases; Mouquin and Macrez (1947) in a series of 124 cases of complete heart block, found normal ventricular complexes in only 25 instances. Graybiel and Sprague (1933) record 17 cases of complete A-V block in 395 cases of B.B. block, and Freund and Sokolov (1939) found complete A-V block in 5 per cent of 210 cases of B.B. block. It is also recognized that the form of QRS during complete A-V block may differ from that recorded during sinus rhythm in the same case.

For purposes of investigating the form of the ventricular complexes, a series of 100 cases of complete A-V block has been assembled, comprising patients seen at the National Heart Hospital during the last few years, and others seen at the Middlesex Hospital or in practice by Dr. Evan Bedford, at whose suggestion this investigation was undertaken. The cases were unselected and as far as possible consecutive from both sources. Fifteen patients have been under personal observation during the past year.

## ÆTIOLOGY

The ætiology of the heart block in this series of 100 cases is shown in Table I.

The cases of uncertain ætiology included five patients with a history of "heart trouble" in childhood, six with a history of rheumatic fever but without clinical evidence of rheumatic valvulitis, four in whom there was a possible cardiac infarction, two with a family history of complete heart block, and two in whom the block followed surgical operation.

TABLE I

ÆTIOLOGY AND SEX INCIDENCE

| | Number of cases | Male | Female |
|---|---|---|---|
| Coronary heart disease: | | | |
| (a) Chronic | 43 | 30 | 13 |
| (b) Associated with recent cardiac infarction | 9 | 8 | 1 |
| Congenital | 20 | 9 | 11 |
| Rheumatic heart disease | 3 | 2 | 1 |
| Diphtheric heart disease | 2 | 1 | 1 |
| Syphilis | 1 | 1 | — |
| Pneumonia | 1 | 1 | — |
| Associated with neurological disease | 2 | 2 | — |
| Ætiology uncertain | 19 | 7 | 12 |
| Total | 100 | 61 | 39 |

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There were eight cases in which complete A-V block was transient. In seven of them there was associated coronary heart disease, one having recent cardiac infarction, and the other case was of doubtful ætiology. In only two of these cases was the P–R interval prolonged during the phase of sinus rhythm.

### The Ventricular Complexes

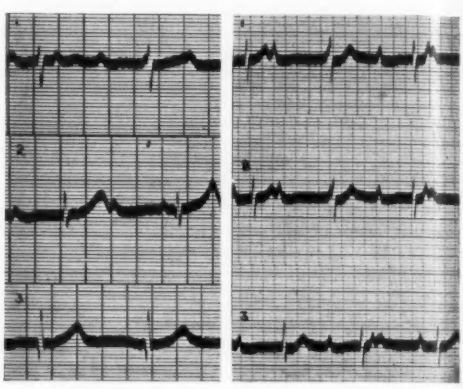
The ventricular complexes have been classified into three groups: (1) Supraventricular pattern. (2) Bundle branch block pattern. (3) Varying complexes.

#### (1) Supraventricular Pattern

Ventricular complexes of normal duration, 0·06–0·10 sec., and with normal S–T and T segments, including cases in which axis deviation was the only abnormality, occurred in 47 of the 100 cases. Of these 47 cases, 19 were congenital, 11 had coronary heart disease, 4 followed rheumatic fever or diphtheria, 1 was associated with neurological disease, 1 followed pneumonia, and 11 were of doubtful ætiology; of this latter group, 5 had a previous history of rheumatic fever and 4 gave a history of heart trouble in early life. Fig. 1 shows the electrocardiograms of two cases of congenital A-V block, with QRS–T complexes of supraventricular pattern.

In addition, in 6 patients with coronary heart disease, the electrocardiograms showed left ventricular preponderance, with QRS complexes of normal duration. Four of them had associated hypertension with systolic blood pressure greater than 200 mm., the blood pressure in the other cases being 170/90 and 180/70 mm. of mercury.

The pulse rate in these cases with normal ventricular complexes varied between 25 and 60 a minute. As shown in Table II, in most of them the rate was relatively high, between 36 and 60 a minute, especially in the cases of congenital heart block, whereas in cases with widened ventricular complexes the rate was slower, between 26 and 40 a minute. The readings recorded in Table II are the average pulse rates, but in individual patients the rate actually varied when observed over a period ; for instance, with an average rate of 30 beats a minute, the rates actually recorded varied between 26 and 34.



A B

Fig. 1.—Complete A-V block with normal QRS complexes.
(A) From a baby six days old; ventricular rate 75 a minute.
(B) From a man aged 18 years; ventricular rate 42 a minute.

The ventricular complexes of complete heart block are usually of supraventricular pattern when the pacemaker is situated in the main bundle and the bundle branches function normally. This is the rule in congenital heart block, and all but one of the 20 cases in this series classed as congenital showed normal QRS complexes, whereas in the cases with coronary heart disease normal QRS complexes occurred in only one-third.

Supraventricular QRS complexes may also occur in complete heart block due to lesions of both

TABLE II
Average Pulse Rate

| Pulse rate per minute | 21–25 | 26–30 | 31–35 | 36–40 | 41–45 | 46–50 | 51–55 | 56–60 |
|---|---|---|---|---|---|---|---|---|
| Congenital: | | | | | | | | |
| Normal complexes .. .. .. | — | 1 | 1 | — | 5 | 4 | 6 | 2 |
| Widened complexes .. .. .. | — | — | — | — | 1 | — | — | — |
| Coronary heart disease (chronic): | | | | | | | | |
| Normal complexes .. .. .. | 1 | — | — | 3 | 4 | 4 | 0 | 3 |
| Widened complexes .. .. .. | 2 | 5 | 7 | 7 | 2 | 5 | 0 | 1 |
| Total cases: | | | | | | | | |
| Normal complexes .. .. .. | 1 | 3 | 3 | 8 | 15 | 9 | 7 | 7 |
| Widened complexes .. .. .. | 3 | 8 | 13 | 14 | 3 | 5 | — | 1 |

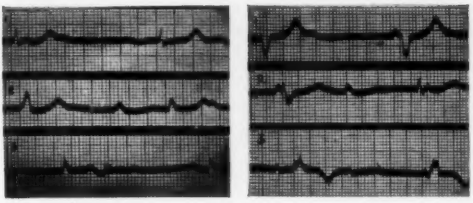
bundle branches (illustrated schematically in Fig. 5A). Wilson and Hermann (1921) found that, in experimental section of both bundle branches, the ventricular complexes might be of the form indicating a right-sided pacemaker, of the form indicating a left-sided pacemaker, or might be of relatively normal outline. To explain the occurrence of relatively normal QRS complexes, they postulated the existence of two pacemakers, one in each bundle branch, acting synchronously, the summation of a lævocardiogram and dextrocardiogram resulting in a ventricular complex of normal outline and duration. In support of this view is the fact that cardiograms showing a change in site of the pacemaker from one side to the other also show transitional forms of QRS complex resembling the normal supraventricular pattern. Cases of complete heart block in which these changes were demonstrated have been reported by Gilchrist and Cohn (1928), and by Bain (1941).

As an alternative explanation, Yater (1936) believes that there is probably a single pacemaker in one or the other bundle branch, sending impulses directly through the interventricular septum into the Purkinje network of the contralateral, as well as into that of the homolateral ventricle.

Mahaim (1931) and Yater (1936) describe cases of complete A-V block in which detailed histological examination of the conducting system showed lesions confined to the bundle branches, and a normal appearance of the A-V node and the main bundle. According to Mahaim (1932), the two branches of the bundle of His are not isolated in their course along the interventricular septum, but are connected by branches as high as the top level of the musculature. The common trunk of the bundle of His is likewise connected with the ventricular myocardium. Because of these high connections, auriculo-ventricular conduction may be normal in spite of destruction of the branches—"bilateral missed block."

In interpreting electrocardiograms of complete A-V block exhibiting QRS complexes of supraventricular pattern, the following points may be considered in favour of bilateral B.B. block as the underlying mechanism: (a) heart disease of coronary ætiology, (b) a relatively slow inherent rate of the pacemaker, (c) the association of supraventricular QRS complexes with those of widened type, or the occurrence of periods of sinus rhythm with B.B. block, especially when the ventricular complexes alternate between right and left branch block.

In two cases of this group widened QRS complexes changed to QRS of normal duration, without any alteration in the pulse rate, which was slow, namely 30 to 36 a minute in both cases (Fig. 2).



A B

FIG. 2.—Complete A-V block with variation in QRS complexes in a man aged 51.
(A) On 24/8/42 shows QRS complexes of normal duration, rate 36 a minute.
(B) On 27/8/42 shows widened QRS complexes, rate 34 a minute.

In another case with widened QRS complexes, the pulse rate was 28 a minute, but increased to 40 a minute when the QRS complexes became normal in duration.

### (2) VENTRICULAR COMPLEXES OF BUNDLE BRANCH BLOCK PATTERN

The B.B. block pattern of ventricular complex is characterized by axis deviation, widening of the QRS, and T waves directed oppositely to the main deflection of QRS. Wilson *et al.* (1944) consider that when QRS has a duration of 0·12 sec. or more and, in lead I, is monophasic and consists of a broad, slurred, flat-topped or bifid R deflection, the præcordial curves almost always show left B.B. block. But when QRS is similarly widened and, in lead I, is biphasic or triphasic and tends to a broad slurred or notched S deflection, the præcordial curves are mainly characteristic of right B.B. block. The occasional exceptions to this general rule may be due to (a) a vertical position of heart, when limb leads suggest right B.B. block and præcordial leads left B.B. block, and vice versa; (b) arborization block, most often seen with extensive myocardial infarction, and due to general depression of conductivity of Purkinje tissue or to local lesions involving the subendocardial Purkinje network over a considerable area. In some cases there is no obvious relation between the form of QRS in præcordial leads and that in limb leads; in these the position of the heart must be classed as indeterminate.

In the present series, 47 per cent of cases showed widened ventricular complexes, with a QRS interval of 0·12 seconds or more, in the presence of complete heart block (see Table III). In the earlier cases, præcordial leads were not available to confirm the type of B.B. block.

(a) In 29 cases the ventricular complexes were of

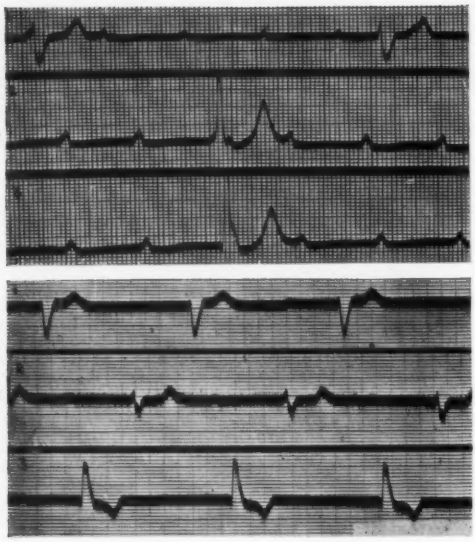



FIG. 3.—Complete A-V block with widened QRS complexes.
(A) QRS complexes 0·14 sec. duration and of right B.B. block pattern (Wilson type).
(B) QRS complexes 0·14 sec. duration and of right B.B. block pattern.

right B.B. block type, 23 being of the Wilson or S type, and 6 of the uncommon type (Fig. 3). Of these, 20 had coronary heart disease, 5 had associated cardiac infarction, 1 was in association with neurological disease, and 3 were of doubtful ætiology.

TABLE III

THE TYPE OF VENTRICULAR COMPLEX IN CASES WITH WIDENED QRS

| | | |
|---|---|---|
| Right bundle branch block. | (a) S type .. | 23 |
| | (b) Uncommon type | 6 |
| Left bundle branch block. | Common type .. | 6 |
| Concordant pattern .. .. .. .. | | 2 |
| Both right and left bundle branch block .. | | 4 |
| Varying complexes .. .. .. .. .. | | 6 |
| | Total .. .. | 47 |

(b) In 6 cases the ventricular complexes were of left B.B. block type. Of these, 3 had coronary heart disease, 1 had congenital heart block, 1 was associated with rheumatic aortic and mitral valvular disease as confirmed by necropsy, and 1 was of doubtful ætiology.

(c) In 2 cases the ventricular complexes were of the concordant type, one of the common and one of the uncommon type. No præcordial leads had been recorded, so that further elucidation of the block was impossible.

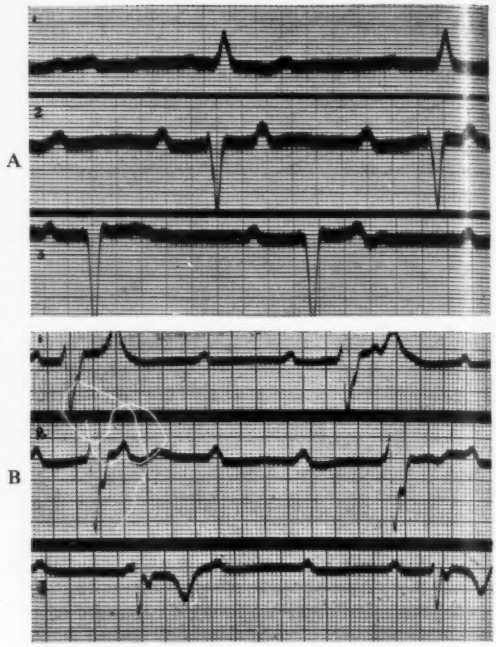



FIG. 4.—Complete A-V block with variation of QRS complexes in a man aged 71.
(A) On 27/7/42 the complexes were of left B.B. block pattern.
(B) On 3/9/42 they were of concordant right B.B. block pattern.

(d) In 4 cases, observed for periods of 1–5 years, the site of the pacemaker varied between left and right ventricle. Of these, one had coronary heart disease and the others were of uncertain ætiology. An example of the cardiograms in this group is given in Fig. 4, which shows (a) QRS complexes of left B.B. block pattern, rate 34 a minute, and (b), four weeks later, QRS complexes of concordant right B.B. block pattern, rate 25 a minute.

(e) Finally, in 6 cases, the widened ventricular complexes assumed varying patterns in the same tracings.

## THE RELATION OF BUNDLE BRANCH BLOCK TO COMPLETE A-V BLOCK

The original interpretation of the B.B. block pattern of QRS, based on experiments in dogs, has in recent years been reversed, largely by the work of Wilson and his associates. Histological investigation of the bundle system in diseased hearts has failed to supply convincing proof of either the

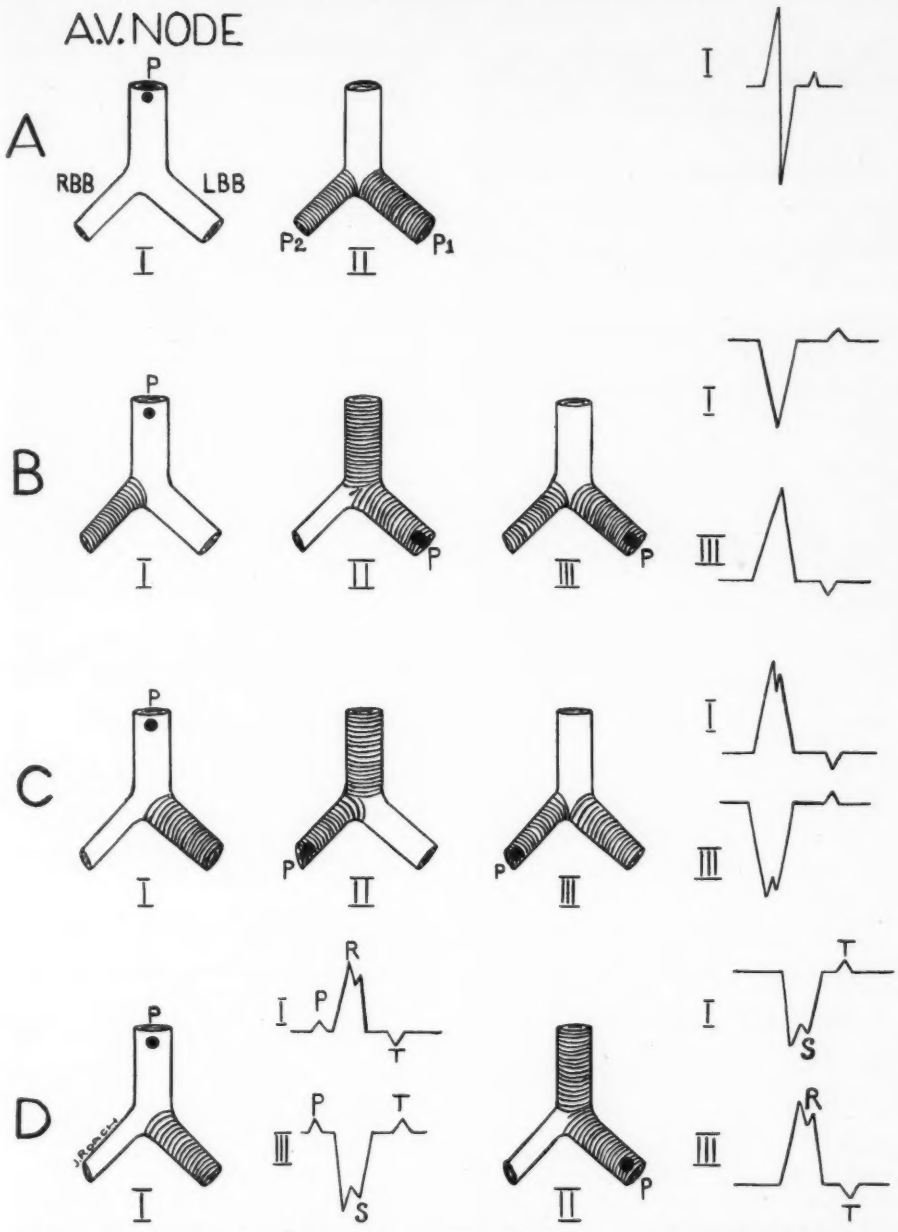



FIG. 5.—Diagram of bundle showing location of ventricular pacemaker (P) site of lesion (shaded zone), and ventricular complexes of electrocardiogram. R.B.B.: right bundle branch. L.B.B.: left bundle branch.

(A) Production of QRS complexes of normal duration. P 1 and P 2 are simultaneous pacemakers in right and left ventricle.

(B) and (C). Production of widened ventricular complexes.

(D) (I) Sinus rhythm; ventricular pacemaker in A-V node; lesion involves the left branch of bundle; cardiogram shows left B.B. block complex.

(II) Complete A-V block; ventricular pacemaker in left branch distal to lesion, now involving also main stem of bundle; cardiogram shows right B.B. block complex.

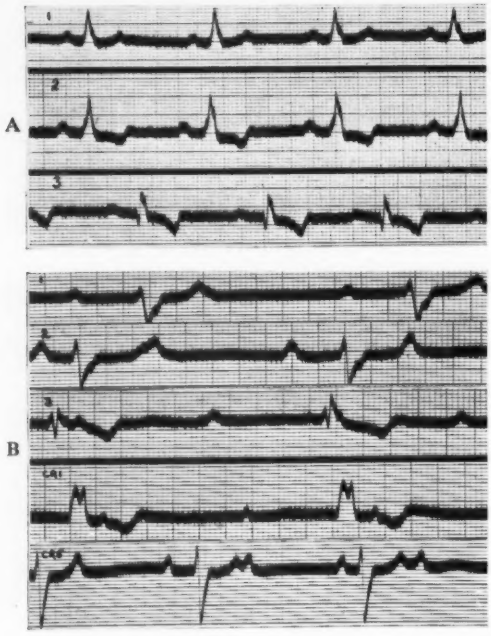



FIG. 6.—Alteration of QRS complexes with change in rhythm.

(A) On 10/1/45, sinus rhythm, rate 50 a minute, P–R interval normal, and QRS complexes of left B.B. block pattern.

(B) On 7/8/47, complete A-V block, rate 30 a minute, and QRS complexes of right B.B. block.

This patient, a woman aged 32, died suddenly on 22/8/47.

old or the new view. Master *et al.* (1940) believe that the B.B. block curve is more often due to diffuse myocardial damage associated with great enlargement of the corresponding ventricle than to a localized lesion of a bundle branch, and Rasmussen and Thingstead (1939) have supported a similar view.

When a lesion of the ventricular septum involves the main stem of the bundle it is likely to extend to the bundle branches and, therefore, we should expect to find complete A-V block sometimes associated with B.B. block in the cardiogram. In fact, QRS of B.B. block pattern occurred in 47 per cent of the present cases of complete block. We may consider this association in relation to the pathogenesis of the B.B. block cardiogram. It is usually assumed that ventricular complexes of, say, right B.B. block pattern indicate defective conduction in the right branch of the bundle, but an alternative explanation is to assume that the ventricular pacemaker is located in the left branch of the bundle. This is illustrated schematically in Fig. 5 (B and C).

Variations in the form of QRS may accompany the change from sinus rhythm to complete heart block, or vice versa, and these are of particular interest in regard to locating the ventricular pacemaker and to fixing the site of the bundle lesion. In 6 cases where records of both sinus rhythm and of complete A-V block were obtained, significant changes in the form of QRS occurred. In five of them, complete A-V block was combined with QRS of right B.B. block pattern, and sinus rhythm with a QRS of left B.B. block pattern (see Fig. 6). This may be explained by supposing that there is a lesion of the left branch and that during sinus rhythm the right branch only conducts. When the lesion extends from the left branch to the main stem of the bundle, causing complete A-V block, the ventricular pacemaker shifts to the left branch distal to the site of the lesion, thus producing right B.B. block pattern in the cardiogram. This hypothesis is illustrated schematically in Fig. 5 (D). Fig. 7 shows the cardiograms of a man aged 54; during sinus

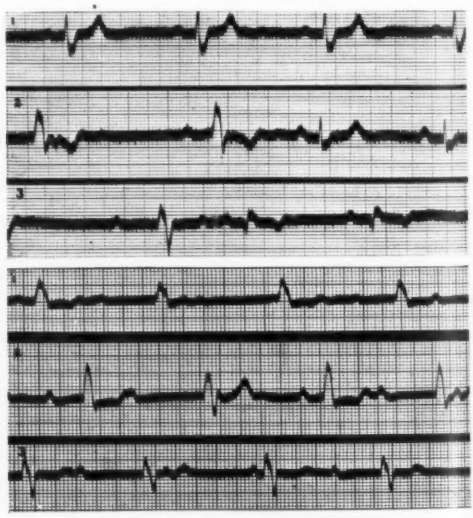



FIG. 7.—(A) Electrocardiogram on 5/10/43, shows in lead I, and latter part of leads II and III, sinus rhythm, rate 48 a minute, and normal P R interval; the QRS complexes are of the Wilson type of B.B. block. In the first half of leads II and III there is complete A-V block, rate 34 a minute, QRS complexes being of left B.B. block pattern.

(B) On 14/10/43, shows complete A-V block, with QRS complexes of left B.B. pattern.

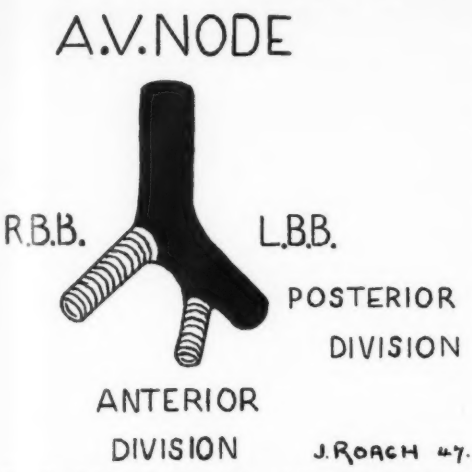



FIG. 8.—Schematic illustration of the blood supply of the junctional tissues. The area supplied by the posterior perforating branches is shown in black, and the shaded area represents the area supplied by the anterior branches.

rhythm the QRS complexes are of right B.B. block pattern and during complete A-V block, of left B.B. block pattern.

### THE BLOOD SUPPLY TO THE BUNDLE

Gross (1921) describes the blood supply to the A-V node and the main bundle as being mainly from a special artery, the ramus septi fibrosi arising from the posterior interventricular artery, which is a branch of the right coronary artery in 90 per cent of hearts; the posterior division of the left bundle branch is also supplied from the same source. The right bundle branch and the anterior division of the left bundle branch are supplied mainly by anterior perforating septal branches of the anterior interventricular artery, a branch of the left coronary artery; the terminal part the right bundle branch may receive some supply from the posterior perforating arteries. The blood supply of the junctional tissues is illustrated schematically in Fig. 8. In normal hearts, the anastomoses between the anterior and posterior perforating septal arteries are very fine, but with the slow obliteration of these arteries by coronary vascular disease, the anastomoses undoubtedly become more effective. Blumgart *et al.* (1940) consider that, irrespective of age, anastomoses measuring 40 micra or more in diameter between the right and left coronary arteries rarely exist in the absence of partial or complete arterial occlusion. Permanent complete A-V block is usually associated with slow and progressive diminution of the blood supply of the posterior portion of the heart, which is through the posterior perforating septal arteries, and it has been shown that these vessels, besides supplying the main bundle, also supply the posterior division of the left bundle branch. The common occurrence of QRS of right B.B. block pattern in complete heart block has been noted; in 5 such cases there were intervals of sinus rhythm with left B.B. block. It is postulated that, with gradual diminution of blood supply, there is functional block of part of the left bundle branch and eventually of the main bundle and that, with the occurrence of complete A-V block, the new pacemaker is situated in the area of irritable tissue below the block, giving rise to a right B.B. block pattern of ventricular complex.

### VARYING VENTRICULAR COMPLEXES

The appearance of varying ventricular complexes in complete heart block may be associated with

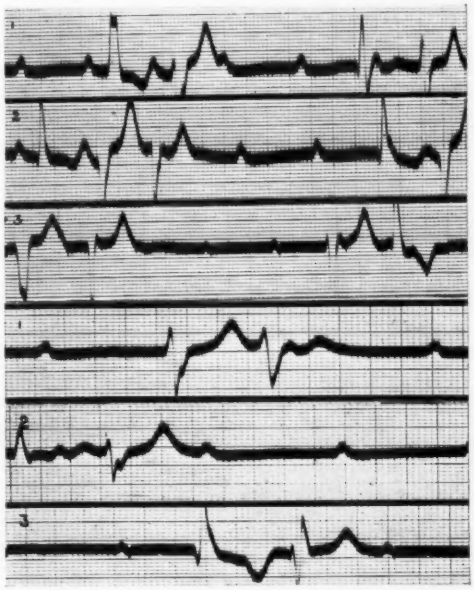



FIG. 9.—Complete A-V block with varying ventricular complexes in the same tracing.

(A) On 19/1/44, shows widened QRS complexes varying in shape in lead I, and multiple extrasystoles producing coupling.

(B) On 18/4/45, showing QRS complexes of right B.B. block pattern. Coupling due to extrasystoles still persists.

This patient, a woman aged 47, was seen again in July 1947; the ætiology of the heart block was unknown.

(a) changing site of the pacemaker; (b) the conduction of occasional auricular impulses; or (c) the occurrence of ventricular extrasystoles.

(a) *Changing site of the pacemaker.* There were six cases in which the ventricular complexes varied in the same tracing (see Fig. 9), indicating either a changing site of the pacemaker, or a varying path of the impulses. In only one of them were transitional complexes between the dextro- and lævo-cardiogram types detected. Five of the cases had coronary heart disease, and the other was of uncertain ætiology. As already described under group 2, there were also 4 cases in which, during observation over a period, the complexes varied between left B.B. block and right B.B. block pattern (see Fig. 4).

(b) *The conduction of auricular impulses* was clearly responsible for variation in the ventricular complexes in records of only three cases (Fig. 7). In one, each of the left B.B. block complexes was closely related to a P wave, although the P–R interval was variable, i.e. 0·24 to 0·36 seconds (Fig. 10).

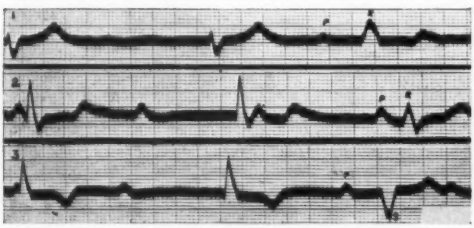


FIG. 10.—Variation in the QRS complexes due to conducted auricular impulses in which the P–R interval varies.

(c) *Ventricular extrasystoles* were recorded in 20 cases, originating in the right ventricle in 7, in the left in 8, and from both ventricles in 5 cases. When ventricular extrasystoles occur in association with widened ventricular complexes, the site of origin of the extrasystoles is usually in the same ventricle as the pacemaker. Thus there were 6 cases with a left-sided pacemaker and extrasystoles, and in 5 of them the extrasystoles arose in the left ventricle (Fig. 11). In one case with a right-sided pacemaker extrasystoles arose in both ventricles. Coupling due to ventricular extrasystoles was recorded in 6 cases of which only one was receiving digitalis. When complexes suggesting both left and right-sided pacemakers are seen, it is necessary to consider the possibility of intermittent auriculo-ventricular conduction, especially if there are no transitional complexes. In this case, the conducted ventricular complex bears a certain relation to the P wave although the P–R interval may show some variation. According to Katz, this is a more common cause of variation in form of the QRS complexes than is the occurrence of extrasystoles. Fig. 12 shows a tracing in which, in lead III, the second and third complexes are definitely extrasystolic, as they bear no relation to P waves.

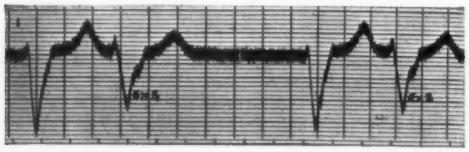


FIG. 11.—Complete A-V block, with ventricular extrasystoles resembling the main complexes.

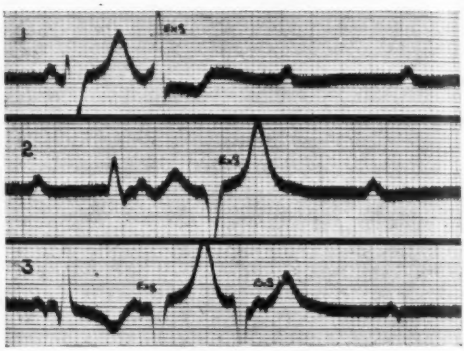


FIG. 12.—The second and third complexes in lead III of this tracing are ventricular extrasystoles, bearing no relation to P waves.

When varying complexes, due to a changing site of pacemaker, accompany complete heart block, the lesions are more likely to be found in the bundle branches than in the main bundle, according to the work of Wilson and Hermann, though destructive lesions of the terminal portion of the main bundle were found by Don, Grant, and Camp (1932).

As already stated, to explain the appearance of variable complexes, Wilson and Hermann (1921), and Gilchrist and Cohn (1928) postulate the existence of two pacemakers, with a change in leadership from one to the other. Yater *et al.* (1936), while accepting this concept, also consider that the site of the pacemaker may change from time to time in the same bundle branch and, in addition, that the path of the excitation wave may vary after leaving the pacemaker. Clinically, bilateral bundle branch lesions can only be diagnosed with certainty when there are intervals of sinus rhythm with alternate right and left B.B. block complexes.

### The Ætiology of Complete Heart Block in Relation to the Form of the QRS Complexes

(a) *Congenital Heart Block.* According to Yater (1929), complete heart block may be regarded as congenital when a slow pulse has been found at an early age, and when there is no history of infection such as diphtheria, rheumatic fever, chorea, or congenital syphilis. A history of syncopal attacks early in life, though rare, and the presence of signs of congenital heart disease, are supporting evidence of a congenital ætiology.

In 20 cases of this series, heart block was regarded as being congenital on the grounds of its recognition in childhood or adolescence, of the absence of any relevant history of infection, and, in four cases, of the presence of signs of congenital heart disease. In eleven of them, electrocardiograms showed QRS complexes of characteristically normal supraventricular form and of short duration (0·06–0·08 sec.) without any axis deviation. In the four cases with associated congenital heart disease, there was right axis deviation with QRS complexes of normal duration. Four cases showed somewhat wider QRS complexes (0·08–0·1 sec.) which were slightly notched, and one case showed widened QRS complexes (0·12 sec.) of left B.B. block pattern. Thus in congenital heart block the QRS complexes are commonly of supraventricular type, and the ventricular rate is relatively rapid, between 40 and 60 a minute. Similar findings have been reported by Leech (1930). On this evidence, we may assume that, in congenital complete block, the lesion usually involves the main bundle.

(b) *Cardiac Infarction.* There were nine cases of complete A-V block associated with clinical evidence of cardiac infarction and six of them showed QRS complexes of right B.B. block type. (1) In three cases definite cardiographic changes indicative of acute infarction were recorded; it was posterior in two (see Fig. 13) and anterior in the other. (2) In one case progression from partial to complete A-V block was recorded and the T waves in leads II and III were observed to become inverted and later upright again; the QRS complexes were of normal duration. (3) In the remaining five cases there was a reliable history of cardiac infarction but no definite electrocardiographic evidence of it. All of them exhibited widened ventricular complexes which were of right B.B. block pattern in four, and of concordant uncommon B.B. block pattern in the other.

Whilst heart block is common with gradual ischæmia of the junctional tissues, it is uncommon in cardiac infarction in which it is more often temporary, lasting hours or days, than permanent. Levine found only two cases of complete block in a series of 145 cases of cardiac infarction with autopsy control; Schwartz (1936) described 15 cases, and Cookson (1942) described 5 cases in a review of 200 cases of cardiac infarction. Complete heart block is usually associated with posterior infarction involving the septum when, as shown in this series, it is often associated with QRS complexes of right B.B. block pattern. Antero-septal infarcts are rarely associated with complete heart block unless the infarct extends to the upper part of the septum.

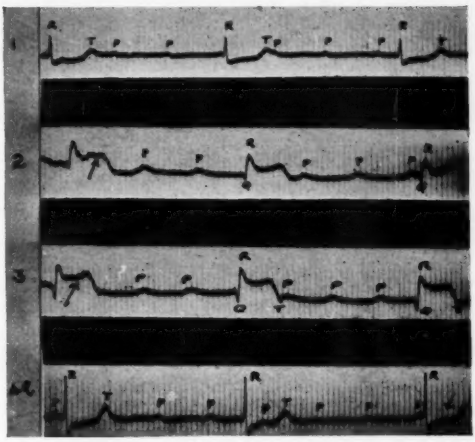


Fig. 13.—Complete A-V block, rate 37 a minute, associated with recent posterior cardiac infarction in a man aged 65, who suddenly collapsed with præcordial pain and dyspnœa. The electrocardiogram was recorded several hours later. (Time marker 0·05 sec.)

In the presence of bundle branch lesions, the electrocardiographic changes of cardiac infarction may be obscured, especially in the case of left B.B. block. Wilson *et al.* (1944) have found that right B.B. block does not obscure the changes characteristic of posterior infarction in the limb leads, though it may cause inversion of the T waves in right-sided præcordial leads and occasionally in lead III (Wolferth and Levezey, 1947). The cardiogram in Fig. 14, from a patient giving a history of cardiac infarction, shows complete A-V block with QRS complexes of right B.B. block pattern, and sharply inverted T waves in leads CR 3 and CR 4, suggesting anterior septal damage.

### The Prognostic Significance of the QRS Pattern

In cases of complete A-V block exhibiting normal QRS complexes, in which the lesion is located high up in the bundle stem and is often congenital in

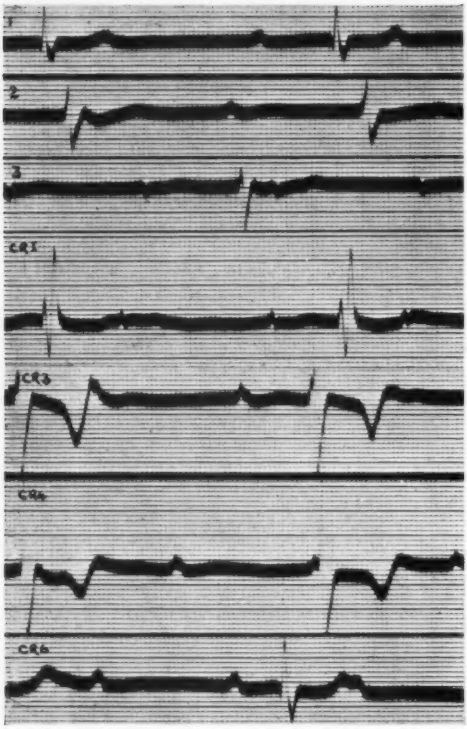



FIG. 14.—Complete A-V block, rate 25 a minute, in a man aged 43, with a history of cardiac infarction. The limb leads show QRS of right B.B. block pattern; leads CR 3 and CR 4 show sharp deeply inverted T waves, suggesting anterior septal damage.

origin, the heart rate is faster and the liability to Adams-Stokes attacks is far less than in cases exhibiting widened QRS complexes, in which the lesion involves the bundle branches, often due to coronary disease, and the heart rate is slower. Thus in my 53 cases with normal QRS complexes, only 20 (38 per cent) were recorded as having Adams-Stokes attacks compared with 31 (66 per cent) of the 47 cases with widened QRS complexes.

Katz considers that the expectation of life is seldom longer than a year or two in complete block with widened QRS complexes. Three of my cases of this type died suddenly before the age of 40, though four others had been under observation for a period of five years. In general terms, however, the prognosis in complete A-V block may be assessed on the basis of the underlying heart disease, being relatively good when the ætiology is congenital and more serious when it is coronary.

## SUMMARY

A series of 100 cases of complete heart block has been investigated with special regard to the form of the ventricular complexes exhibited in electrocardiograms. The most frequent ætiological cause of complete heart block was coronary heart disease which occurred in 52 cases, and next a congenital origin which was probable in 20 cases.

The ventricular complexes found in complete heart block may be classified as follows:—

(1) *Supraventricular pattern* (a) due to block in the main bundle stem, (b) occasionally due to bilateral bundle branch block.

(2) *Bundle branch block pattern* (a) due to block in a bundle branch, (b) due to block in the main stem associated with bundle branch block.

(3) *Varying pattern* (a) due to shifting pacemaker, (b) due to conducted auricular beats, (c) due to extrasystoles.

In coronary heart disease, complete heart block is usually associated with obstruction of the posterior blood supply, and occurs more often with posterior than with anterior cardiac infarction. The frequency of associated right B.B. block in coronary disease can be explained in terms of the blood supply to the conducting system. In congenital complete heart block, the QRS complexes were of supraventricular pattern in 19 out of 20 cases.

In complete block exhibiting QRS complexes of B.B. block pattern, the pacemaker is situated lower in the bundle, its inherent rate is slower, Adams-Stokes attacks occur more often, and the expectation of life is less than in cases of complete block exhibiting normal ventricular complexes.

I have to thank members of the staff of the National Heart Hospital for permission to publish records of their cases, and Dr. Evan Bedford for putting at my disposal cases seen at the Middlesex Hospital and in practice.

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# THE REGENERATIVE CAPACITY OF MAMMALIAN HEART MUSCLE

BY

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King in 1940, in this journal, described appearances that he believed to be those of myocardial regeneration in a case where death had occurred four days after a stab wound of the chest. A review of the literature, however, shows the opinion most generally held is that hyperplasia of cardiac muscle does not occur. Another consideration prompted the present study: Le Gros Clark (1946) showed that considerable regeneration of skeletal muscle took place in the rabbit following experimental injury. It therefore seemed desirable that the regenerative power of the rabbit myocardium should be studied.

## Methods and Results

It is very difficult to produce by crushing comparable lesions in rapidly beating hearts, and as burning had been employed successfully by Thomas and Harrison (1944) for functional recovery tests in rats, it was decided to use this type of lesion.

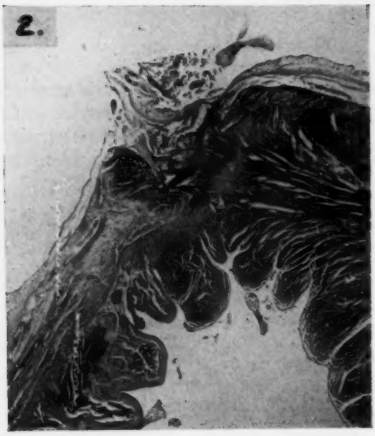
In adult rabbits the heart was exposed under anæsthesia, with full aseptic precautions, and a severe burn made in the lower third of the left ventricle by the application of the head of a nail, 5 mm. in diameter, heated to a dull redness. The chest wound was closed and the animals allowed to survive for periods ranging from 3 days to 3 months. The hearts were then examined microscopically.

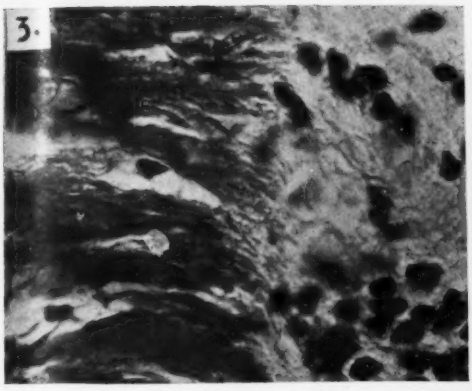
The appearances in the injured area were at first simply those of severe inflammatory reaction with destruction of tissue. Later a progressive organization into scar tissue occurred; but there was no evidence of regeneration of cardiac muscle.

At first the site of injury showed as a deep crater (Fig. 2) filled with fibrin clot and necrotic tissue to which pericardial adhesions had become attached. Surrounding this was an area containing damaged muscle fibres invested by macrophages of different kinds. Amongst these cells monocytes were prominent (Fig. 4), a point which has been noted by pathologists in acute muscle infections generally. The damaged muscle fibres showed fragmentation (Fig. 5), the lines of cleavage seeming to be at the intercalated discs, and their transverse striations and nuclei stained less strongly. Very early on there were great numbers of fibroblasts to be seen within the injured area which was soon invaded by capillaries from the neighbouring healthy tissue (Fig. 1). After a month there was a clear line of demarcation between undamaged muscle and scar tissue (Fig. 3) although here and there fragments of muscle with attendant phagocytes could still be seen. At no stage was sprouting of myoblasts detected: nor were mitotic figures found, although it must be admitted that Le Gros Clark (1946) found nuclear division took place as a general rule by amitosis in regenerating skeletal muscle. The longitudinal splitting of fibres described by King (1940) was not observed.

## Discussion

Compared with skeletal muscle cardiac muscle is much less easy to trace in serial sections, but even so it is felt that no signs of regeneration were missed. The explanation of the difference in regenerative capacity of the two classes of muscle, as shown in the rabbit, may be histological, or it may be that in the heart rest cannot be enjoyed by the damaged fibres. Distinguishing features of cardiac muscle are the ill-defined or absent sarcolemma, centrally placed nuclei, branching fibres, well marked longitudinal striation, granular sarcoplasm which somewhat masks the transverse striations, and the presence of intercalated discs. It is usually stated that there is continuity of myofibrils through the discs. Fig. 6 shows this, but I am not satisfied that such continuity exists through all discs and it may be that the discs, whatever their physiological significance may be, act as an impediment to regeneration. It might be argued that burning is



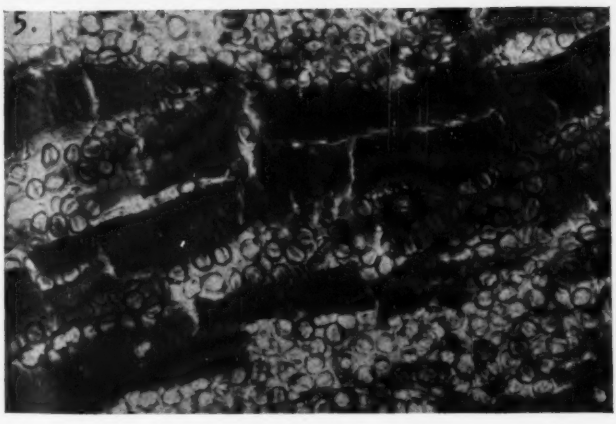


FIG. 1.—Section 1.2.3. of rabbit heart (R.7) three days after operation. Healthy fibres above and to left. Numerous fibroblasts are shown within the damaged area which is becoming revascularized. Magnification: ×500.

FIG. 2.—Section 32.2.3. of rabbit heart (R.4) seven days after operation. A general view of the crater produced by the burn is shown. Magnification: ×7.

FIG. 3.—Section 3.2.2. of rabbit heart (R.10) one month after operation. Healthy tissue to left, scar tissue to right. The line of demarcation is sharp. Magnification: ×650.

FIG. 4.—Section 32.2.3. of rabbit heart (R.4) seven days after operation. Damaged myocardial fibres are shown surrounded by great numbers of phagocytes. A large monocyte is especially pronounced. Magnification: ×530.

FIG. 5.—Section 1.2.3. of rabbit heart (R.7) three days after operation. To show fragmentation of the muscle fibres. Magnification: ×650.

FIG. 6.—Section of undamaged rabbit heart muscle. To show continuity of myofibrils through an intercalated disc. Magnification: ×1375.

too severe a lesion for a fair assessment of results, but Harrison (1947) who carried out similar experiments also showed that skeletal muscle in rabbits did regenerate after burning by diathermy.

SUMMARY

Following experimental injury of the rabbit heart by burning there is no evidence of regeneration of cardiac muscle.

To Professor C. M. West, University College, Cardiff, in whose department this work was started, and to Professor John Kirk, my present chief, I wish to express my gratitude for much helpful advice. The photomicrographs are the work of Mr. S. R. Scarfe.

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# INCIPIENT SYMMETRICAL PERIPHERAL GANGRENE COMPLICATING PAROXYSMAL TACHYCARDIA

BY

D. GORDON ABRAHAMS

Received February 11, 1948

Bilaterally symmetrical incipient gangrene of all the bodily extremities is sufficiently rare to merit the report of the following case.

## Case Report

Mrs. H., a housewife, aged 47 years, was admitted to hospital in January 1947. She had been married 27 years, and had two healthy adult sons. Three days previously she was suddenly taken ill with diarrhœa and sickness, accompanied by faintness and a "fluttering feeling around the heart." Her doctor recommended her admission to hospital because her pulse was extremely rapid, and because her general condition deteriorated swiftly. Her history revealed two similar attacks, the last six weeks previously. This began with temporary loss of consciousness and lasted about two days, and on the second day she noticed that her hands and feet were very cold. However, she soon made an uneventful recovery. For the last year she had been conscious of a fluttering in the chest on exertion, which had lately been lasting longer and had been more easily provoked.

On admission, she was obviously a very ill woman. The hands, feet, nose and ears were very cold, extremely cyanosed, painful, but anæsthetic to light touch. The skin of the extremities did not blanch on pressure. The radial, ulnar, and dorsalis pedis pulses were imperceptible. The brachial and popliteal pulses were palpable. The systolic blood pressure was 65 mm. in the arms, and 85 mm. in the legs; the diastolic was unobtainable. The pulse rate was extremely rapid. Clinically, the heart was enlarged to right and left, but the limits were difficult to define. An apical thrill and murmur were noted, but they could not be timed owing to the tachycardia. The neck veins were markedly distended and

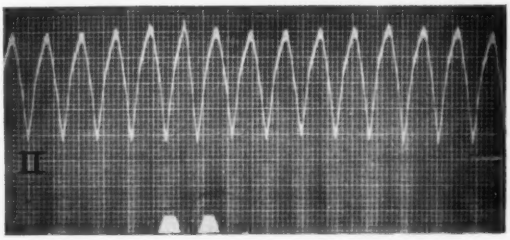


FIG. 1.—Ventricular tachycardia, rate 230 a minute.

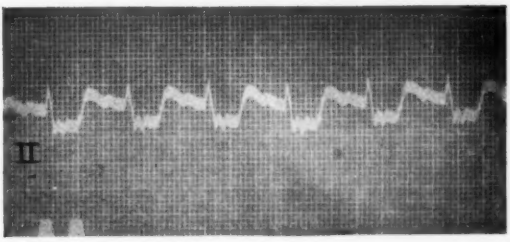


FIG. 2.—Normal rhythm, rate 97, P–R interval 0·16 sec., S–T segment depressed.

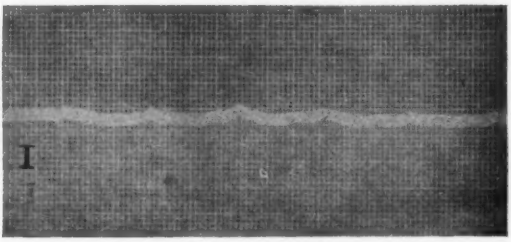


FIG. 3.—Ventricular asystole, ventricular rate 90.

quivering, being engorged up to the ears, whilst the arm veins were so empty of blood that they could not be seen at all. The liver was just palpable; ascites and pitting œdema were absent, but there were a few moist sounds at the lung bases. Throughout all this time the patient remained co-operative, calm, alert, and in full possession of her mental faculties.

A portable chest X-ray provided no useful information. An electrocardiogram showed a regular paroxysmal ventricular tachycardia at a rate of 230 a minute (Fig. 1).

Thus the clinical picture was that of extreme circulatory failure, of which the immediate precipitating cause was the tachycardia. It was imperative to slow the heart rate and to raise the blood pressure as soon as possible, and quinidine was given intravenously for this purpose. After a test dose of 3 grains had excluded idiosyncrasy to the drug, a solution containing one gram of quinidine sulphate in 100 ml. of glucose saline was administered by slow intravenous drip. Half an hour later, when the patient had received approximately 60 ml. of the solution (0·6 g. quinidine), she suddenly became distressed and lost consciousness. The drip was discontinued and a further electrocardiogram was taken (Fig. 2). This was thought to show regular rhythm at a rate of 97 a minute; the QRS complexes were less widened. A period of ventricular asystole then ensued, with an auricular rate of 90 a minute (Fig. 3). This was followed by cardiac standstill and death four hours after her admission.

*Post-mortem examination.* The cardiovascular system alone showed relevant pathological changes. There was extensive syphilitic aortitis, with aneurysmal dilatation of the ascending part and arch of the aorta. The coronary arteries were

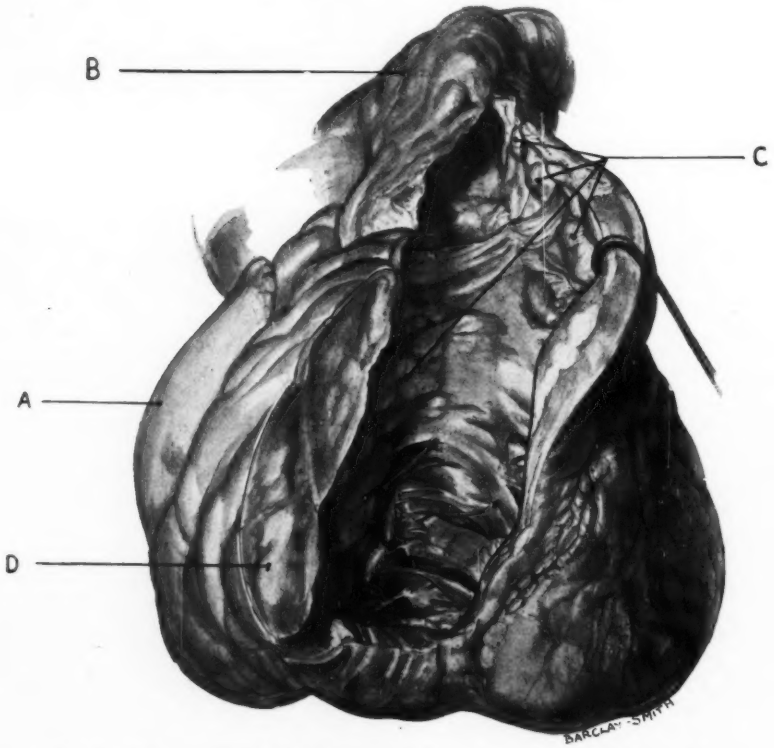


FIG. 4.—Drawing of heart with right ventricular cavity open. (A) Right auricle, (B) Pulmonary artery, (C) Granulomatous masses in right ventricle and pulmonary artery, and (D) Gummatous infiltration of the wall of the right ventricle.

FIG. 5.—Gumma of myocardium. Magnification ×125.

healthy and showed no sign of narrowing of their orifices. The left auricle and ventricle appeared normal, but the muscle of the right ventricle and also the interventricular septum showed diffuse fibrotic infiltration. There were, in addition, numerous large, discrete, granulomatous lesions distributed over the internal surface of the right ventricle; one particular mass lying under the tricuspid valve measured three-quarters of an inch in diameter. The pulmonary artery was similarly affected (Fig. 4). The arteries to the upper and lower extremities showed no abnormality throughout their lengths. Section and microscopic examination revealed that both the diffuse infiltration and the granulomatous masses were syphilitic in nature (Fig. 5 and 6).

## DISCUSSION

The case presents several points of interest. Gumma of the heart is comparatively rare. In fourteen years of extensive study of syphilitic cardiovascular disease, Maynard (1943) stated that this lesion had been encountered only once at necropsy. When present, gummata usually occur in the left ventricular myocardium and particularly in the basal portion of the septum (Sohval, 1935). Sohval also states that diffuse gummatous myocarditis is extremely rare by itself, being usually associated with gumma of the heart, as it was in this case. Syphilis of the pulmonary artery is also rare, though in a review of cases showing aneurysm of the pulmonary artery, Boyd (1941) stated that syphilis was the ætiological factor in thirty-three cases (31 per cent).

A paroxysm of ventricular tachycardia usually lasts no more than an hour (Cooke and White, 1943), but longer attacks, lasting 1 to 3 weeks have been recorded (Dubbs and Parmet, 1942; Beers and de la Chapelle, 1947). In the present case, the paroxysm lasted just over three days.

Symmetrical peripheral gangrene is mentioned by Fishberg (1944) as occurring in occlusion of the mitral valve by a ball thrombus, and in massive cardiac infarction. I can find no other references concerning its occurrence in cardiac infarction, but Abramson (1924) recorded it in a review of ball thrombi of the heart. He stated that severe disturbances of the general circulation were the rule, and that gangrene of the extremities sometimes occurred, which he thought was usually due to peripheral arterial emboli. Tight or button-hole stenosis of the mitral valve frequently gives rise to intense cyanosis, and coldness of the extremities, but not to gangrene.

Fishberg (1938) noted the co-existence of "collapsed" veins in the extremities with engorged jugular veins, in extreme failure of the right heart. This he attributed to compensatory vaso-constriction in the extremities evoked reflexly by extreme diminution in cardiac output. In this connection he observed symmetrical peripheral incipient gangrene on two occasions.

Bruce Perry and Davie (1939) reported bilateral symmetrical peripheral incipient gangrene in the lower limbs in a case of hypertensive heart disease, terminating in congestive failure, and agreed with Fishberg that excessive peripheral vasoconstriction was responsible. Chatterjee (1940) noted peripheral symmetrical incipient gangrene during the course of

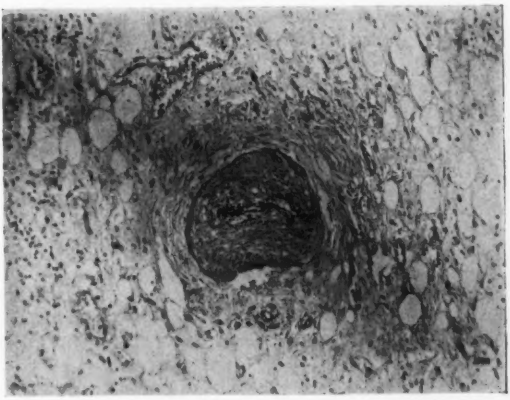

FIG. 6.—Obliterating endarteritis of myocardial vessel. Magnification ×125.

lobar pneumonia, and concluded that it was due to acute circulatory failure, probably reflex in origin.

In the present case, neither arterial emboli, nor luetic arteritis played any local part in the genesis of the gangrene. It seems certain, therefore, that reflex peripheral vasospasm, as indicated by Fishberg, was responsible, and that this was evoked by a critically low cardiac output, due to persistent ventricular tachycardia.

## Summary and Conclusions

A case of incipient symmetrical peripheral gangrene is described, associated with extreme heart failure due to persistent ventricular tachycardia. Necropsy revealed the presence of discrete gummata of the heart and pulmonary artery, and diffuse gummatous infiltration of the right ventricle and interventricular septum. Similar cases are briefly reviewed, and it is concluded that the incipient gangrene was due to compensatory peripheral vasoconstriction.

I wish to thank Dr. J. H. Gubbin and Dr. R. G. M. Longridge for permission to publish this case, and Dr. G. W. D. Henderson who performed the autopsy. I am indebted to Dr. J. W. Shackle for the photomicrographs and to Miss Barclay-Smith for the drawing of the heart. I have to thank Dr. Paul Wood for much helpful criticism in the preparation of this report.

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# TUBERCULOUS PERICARDITIS*

BY

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The incidence of tuberculous pericarditis in this country seems to have been underestimated in the past. The role of tuberculosis as one of the ætiological factors in chronic constrictive pericarditis is being increasingly recognized (White, 1935; Sellors, 1946); yet relatively few studies of the onset or acute stage of the disease have been published, and the majority of these hail from overseas.

## Incidence of Tuberculous Pericarditis

Osler (1893) analysed 1000 autopsies, of which 215 were cases of tuberculosis and 7 had tuberculous pericarditis, giving an incidence of 0·7 per cent in the U.S.A. Norriss (1911), also in the U.S.A., found 1780 cases of tuberculosis in 7219 consecutive autopsies; 82 of the tuberculous cases had pericarditis, an incidence of 1·1 per cent. As compared with these figures Suzman found an incidence of 0·4 per cent in autopsy records at Guy's hospital; 1500 autopsies yielded 102 cases of tuberculosis, of whom 6 had pericarditis. American writers agree in stating that the condition is more frequent in coloured patients than in whites; this, no doubt, has helped to enhance the impression that it is " rare in Europeans " (White, 1935). High susceptibility appears to extend also to coloured African races, for Heimann and Binder (1940) succeeded in collecting 31 examples in Bantu natives in Johannesburg.

Ellman (1945) published a series of 6 cases and suggested that the disease is less infrequent in this country than had hitherto been supposed. Autopsy statistics may be misleading because tuberculous pericarditis sometimes undergoes complete resolution (Ellman, 1945; also Cases 3 and 6 of present series) or because the tuberculous nature of a chronic pericarditis is not always recognizable histologically on examination of excised pericardium or autopsy material. White (1935) had a case in which pericardium excised at operation showed non-specific fibrosis; the patient died shortly after the operation, and at autopsy tubercles were found in the remainder of the pericardium. Dr. T. Semple (personal communication) had a patient with constrictive pericarditis whose excised pericardium was reported histologically as showing non-specific fibrous thickening; four months after the operation the patient developed tuberculosis of an elbow joint. It is certain, therefore, that in addition to the recognized cases of constrictive pericarditis, others are tuberculous in origin despite a non-specific histological picture. Furthermore failure to find tubercle bacilli in a pericardial effusion should not be allowed to cast doubt on a diagnosis of tuberculous pericarditis if there is other evidence, clinical or radiological, pointing to a tuberculous ætiology; Fine and Katz (1944) report an instance of primary tuberculosis of the pericardium in which the pericardial fluid was negative on guinea-pig inoculation; nevertheless the patient died a month later from miliary tuberculosis with meningitis, and tuberculous pericardial thickening was found at autopsy.

Excluding 3 seen in consultation on a single occasion, 8 cases of tuberculous pericarditis have been under my personal observation during the past 13 years; 7 were males; 3 were in hospital during the single year 1947. The incidence would therefore seem to be considerably greater in this country than has previously been suspected. The youngest was an infant of 18 months; three were boys aged 11, 14, and 15; three were men aged 23, 27, and 43; and the woman was aged 20. All writers agree that the incidence is considerably greater in males than in females. Harvey and Whitehill (1937) found 90 per cent males in a series of 95 cases admitted to the Johns Hopkins Hospital during a period of 45 years. Their maximum age incidence was between 20 and 30

* This article includes material used in communications to the British Cardiac Society, May 1948, and the 111 Inter-American Cardiological Congress, June 1948.

(25 per cent of cases), but every decade in life was represented and several patients were over 50.

### Stage at which Pericarditis Develops

Paget (quoted by Ellman, 1945) has stated that most cases arise in the stage of early dissemination following the primary infection. Harvey and Whitehill's findings (1937) are consistent with this view; of their 37 bacteriologically proved cases, only one had previous hæmoptysis. The present series amply bears out Paget's suggestion. Two patients (Cases 1 (Fig. 1–3) and 2 (Fig. 4–6)) developed pericarditis in the presence of an active primary Ghon lesion with hilar gland involvement; one of them had early secondary infiltration as well. Two (Cases 3 (Fig. 7–9) and 7) had pericarditis accompanying an apparently primary tuberculous pleurisy. Three (Cases 4, 6, and 8) occurred in the absence of any demonstrable lung or pleural lesion. In Case 4 the ætiology was obscure at the onset of his acute attack, but a pleural effusion appeared after a month; pericardial constriction followed and the tuberculous ætiology was proved by histological examination of excised pericardium nine months later. Case 6 was never proved tuberculous beyond doubt; but his initial pericarditis was followed by a right-sided pleural effusion after five months, and by a left-sided pleural effusion with recrudescent pericarditis four months later. Case 8 is recent, suspected though as yet unproved, and still under observation. Only once (Case 5) did pericarditis complicate pulmonary disease of longer standing; this was a male, aged 43, with an old calcified lesion at his right apex: he developed a pleural effusion with tubercle bacilli in the fluid and in his sputum, though X-ray failed to reveal signs of activity of his pulmonary lesion; four months later, when he was regarded as convalescent and due for discharge from hospital, a pericardial effusion suddenly developed; the pericardial fluid contained tubercle bacilli.

### Mode of Infection of Pericardium

Riesman (1901) classified tuberculous pericarditis into four groups: (1) with generalized miliary tuberculosis, (2) with serous membrane tuberculosis, (3) by extension from neighbouring foci (regarded by him as the commonest) and (4) primary. Most writers question the existence of anatomically primary tuberculosis of the pericardium. Clinically primary pericardial tuberculosis has been regarded as uncommon in the past; Clarke (1929) stated that careful search revealed only 11 such reported cases in which the diagnosis could not be questioned; Fine and Katz (1944) found only one case reported in children under 15 (by Blatt and Greengard, 1928); they add one of their own. The revival of interest in pericardial constriction, brought about in recent years by the possibilities of surgical treatment, has led to the general belief that clinically primary cases must be less infrequent than these reports would suggest.

No less than three in the present series (Cases 4, 6, and 8) fall into the category of "clinically primary"; yet it seems almost certain that they are, in fact, secondary to tuberculosis of the mediastinal

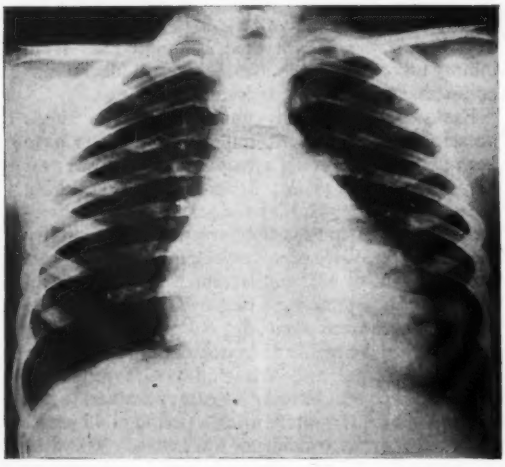

Fig. 1—Case 1. Teleradiogram, 13/12/47. Pericardial effusion with Ghon focus.

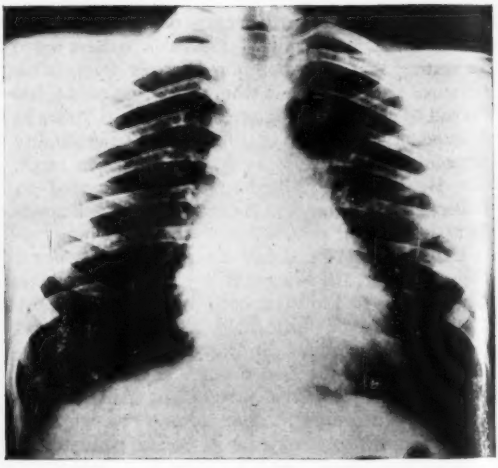

Fig. 2—Case 1. Teleradiogram, 20/1/48. Effusion largely absorbed; Ghon focus persists.

FIG. 3—Case 1. Teleradiograms. Enlarged view of Ghon focus with hilar glands on 13/12/47 and 16/2/48.

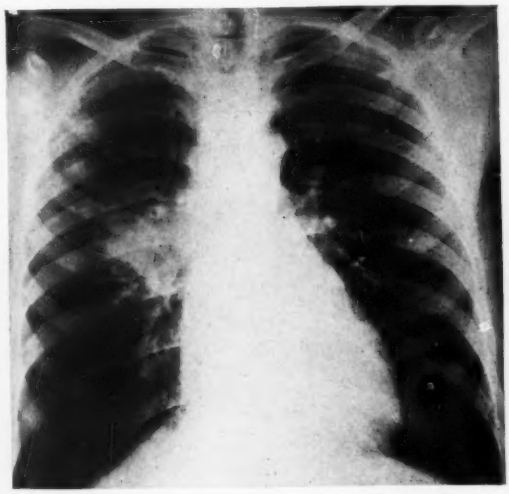

FIG. 4—Case 2. Teleradiogram, 28/11/47. P–A view.

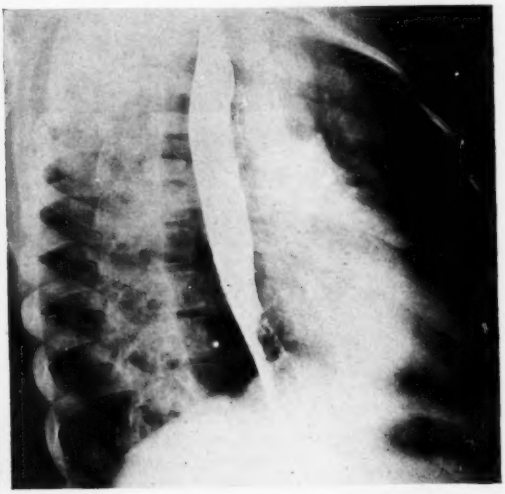

FIG. 5—Case 2. Teleradiogram, 20/1/48. Right oblique view.

lymph glands, from which the pericardium has become infected by extension. Failure to visualize enlarged mediastinal glands radiologically cannot be held to exclude tuberculosis of these glands. In the two cases where pericarditis accompanied an active primary Ghon lesion, the radiological picture strongly suggests direct extension from hilar glands as the probable route of infection (Fig. 3 and 6).

In another patient (Case 3) where pericarditis accompanied a right-sided pleural effusion, serial X-rays revealed transient enlargement of the glands in the left hilum (Fig. 7). Heimann and Binder (1940) found tuberculous mediastinal glands in all their cases but pulmonary lesions were present only in 16 of the 28 autopsies. Harvey and Whitehill (1937) and Hannesson (1941) also regard extension

from mediastinal glands as the most important route of infection.

While miliary tubercles may form in the pericardium during the course of miliary tuberculosis, this form of pericarditis is clinically unimportant; the course of the disease is not modified. It must be borne in mind that tuberculous pericarditis can terminate in miliary tuberculosis; Heimann and Binder (1940) had several cases in which miliary tuberculosis followed tuberculous pericarditis, but they failed to find a single instance in which pericarditis was secondary to miliary tuberculosis; they were dealing with cases of "clinically important" pericarditis.

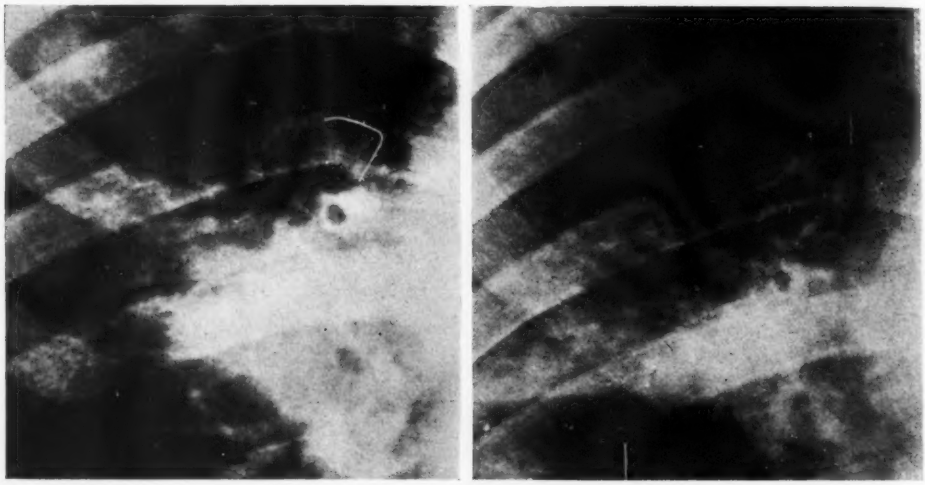

FIG. 6—Case 2. Teleradiograms. Enlarged view of Ghon focus and secondary infiltration on 22/11/47 and 25/4/48.

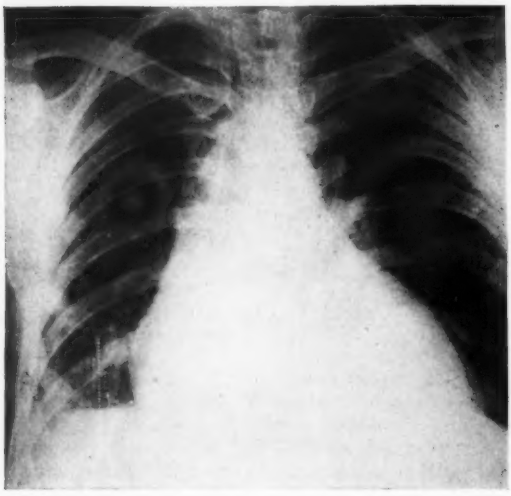

FIG. 7—Case 3. Teleradiogram, 1/5/47, with pericardial and right pleural effusions.

FIG. 8—Case 3. Teleradiogram, 19/5/47, with left hilar enlargement.

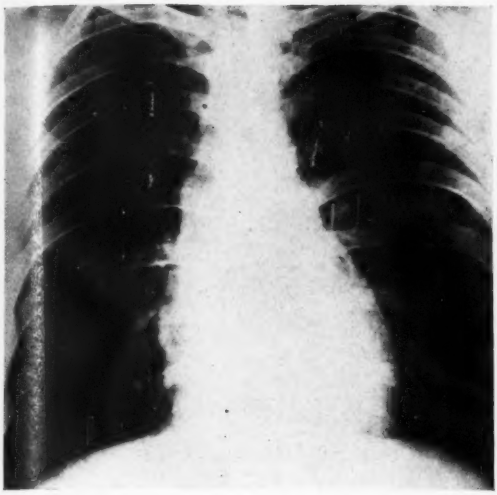

FIG. 9—Case 3. Teleradiogram, 8/9/47, showing complete resolution.

### SYMPTOMATOLOGY

American writers (Harvey and Whitehill, 1937; Hannesson, 1941) differentiate two groups of cases, in one of which the clinical features suggest acute tuberculosis or other acute infection; pericarditis is an incidental finding and is "clinically unimportant." Only one case in the present series falls into this group; this was a young woman of 20, a member of the W.A.A.F. who was admitted to an E.M.S. hospital with fever, tachycardia, flushing, sweating, and signs of a large right-sided pleural effusion from which tubercle bacilli were recovered; there were no cardiac or circulatory symptoms, pericardial friction and effusion being found incidentally. It seems likely that, under conditions obtaining in Great Britain, such cases are more likely to be encountered in fever hospitals or sanatoria than in general hospitals.

The second group is that in which the pericarditis is "clinically important" that is to say the clinical features point to cardiac disease and pericarditis is the presenting symptom. One of the most striking characteristics of this group is the relative insignificance of constitutional symptoms. Almost all writers comment on this (Harvey and Whitehill, 1937; Hannesson, 1941; Suzman, 1943; Barrett and Cole, 1944; Ellman, 1945); and it has been noteworthy in the present series. Loss of weight and night sweats have been conspicuous by their absence. Case 2 was afebrile (Fig. 10); while in Case 3 fever was virtually absent (Fig. 11), a temperature of 99° F. having been recorded only on four occasions during the course of the illness. In the absence of cardiac tamponade the pulse rate rarely exceeds 110 while the sleeping pulse rate has usually been between 80 and 95. Thus Case 1 who had fever (Fig. 12), friction, pericardial effusion, cardiographic evidence of myocarditis, and radiological evidence of an active Ghon lesion, had a sleeping pulse rate between 90 and 95 with a waking pulse rate between 100 and 110; Case 3 with friction and effusion but no fever had a pulse rate between 60 and 70; in Case 2 the pulse rate remained unaltered (sleeping pulse 80, waking pulse 90 to 100) when pericardial friction appeared. In this respect tuberculous pericarditis presents a marked contrast to rheumatic pericarditis. Pallor was present in the four children in the series, the facial appearance being similar to that in rheumatic carditis; but despite the pallor there was remarkable absence of lassitude—the patients being bright, active and alert, almost euphoric in their insistence that they felt nothing wrong at times when they were febrile with friction or signs of effusion. Appetite has generally remained unimpaired.

Subjective symptoms, when present, have consisted of præcordial pain (Case 3), of circulatory embarrasment from cardiac tamponade (Case 5), of pressure cough (Case 1), or have been attributable to a coincident pleurisy (Case 7).

An interesting physical finding has been the persistence of loud pericardial friction over the entire præcordial area in two instances (Cases 1 and 3) in the presence of a moderately large effusion. This feature has previously been noted by Harvey and Whitehill (1937) and by Ellman (1944), though no explanation of such an apparent anomaly has been attempted. In both cases the effusion has seemed of sufficient size to ensure separation of the parietal and visceral layers of the pericardium (see Fig. 1 and 7), and it seems likely that the sound has been produced in the parietal layer itself. In the chronic constrictive stage of tuberculous pericarditis the parietal layer is usually much thickened, often to a considerably greater extent than the visceral layer; it seems reasonable to suppose that this layer will be the seat of inflammatory thickening in the acute stage also. In these circumstances the alternating tension and relaxation which must be communicated to the parietal layer through the inelastic fluid content of the sac at each cardiac diastole and systole, may perhaps be responsible for the production of the friction sound.

### MYOCARDIAL LESIONS

With the exception of Heimann and Binder (1940), who found myocardial lesions in 10 of their 28

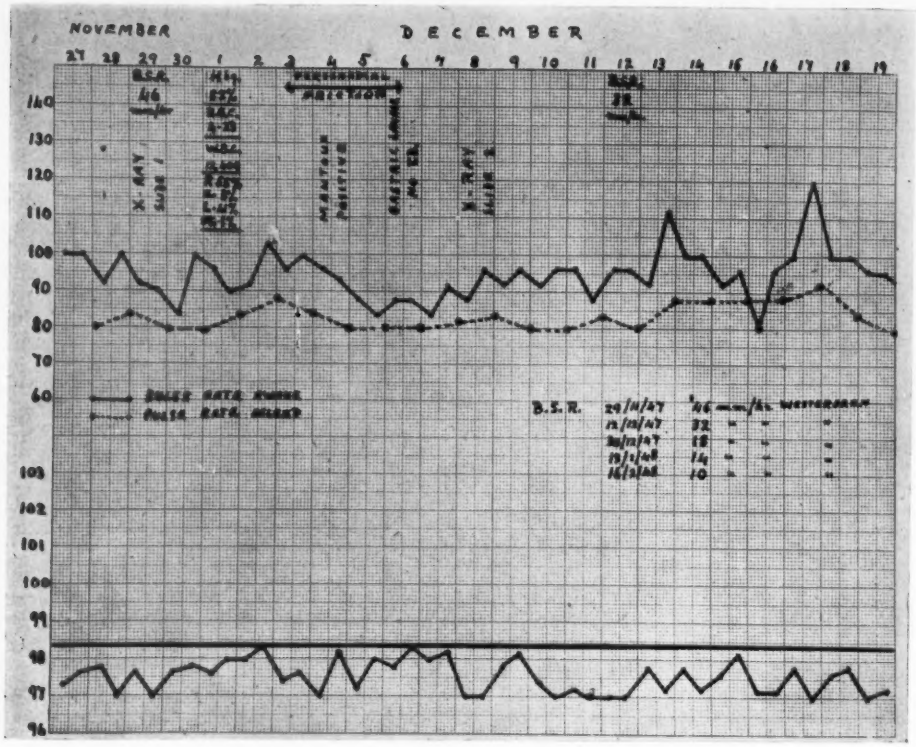


FIG. 10.—Case 2. Temperature and pulse chart.

autopsies in Bantu natives (36 per cent), most writers regard myocardial tuberculosis as rare. Thus Harvey and Whitehill (1937) encountered only two instances of infiltration of the myocardium apart from the presence of miliary tubercles in one patient with miliary tuberculosis; the left auricle was involved in one, the ventricles in the other. One patient in the present series (Case 1) had cardiographic evidence of myocardial involvement; there was M-shaped notching of QRS in lead CR 2 suggesting a lesion of the right bundle branch (Fig. 13): this persisted for a month, then gradually disappeared; in addition, there was T wave inversion in leads I, II, CR 2, CR 4, and later CR 6; this likewise persisted for fully a month after the effusion had completely absorbed.

Harvey and Whitehill found that arrhythmias were confined to patients who had received digitalis; auricular fibrillation occurred in two, latent heart block in two, partial heart block once, complete heart block once, and nodal rhythm once. Case 5 of the present series had auricular fibrillation for a time; he had previously been treated with digitalis and mercurial diuretics. None of the remaining patients received digitalis; and none showed any arrhythmia while under my observation, though one was stated by his doctor to have had cardiac irregularity (the type of which was not specified) at the onset of his illness (Case 6).

### ENDOCARDIAL LESIONS

Tubercles have been found in the endocardium and valves in some recorded cases, but there is no evidence to suggest that chronic valvular disease ever results (Hannesson, 1941); thus tuberculosis was found only once in a series of 300 cases of mitral stenosis. Conversely White states that mitral stenosis is extremely rare in cases of tuberculosis. A previous history of rheumatism was unusual in Harvey and Whitehill's series (1937); three of their patients gave a vague story of joint pains from 6 to 11 months before the onset of their present illness; but the description was not that of a typical rheumatic polyarthritis, and in no instance had there been

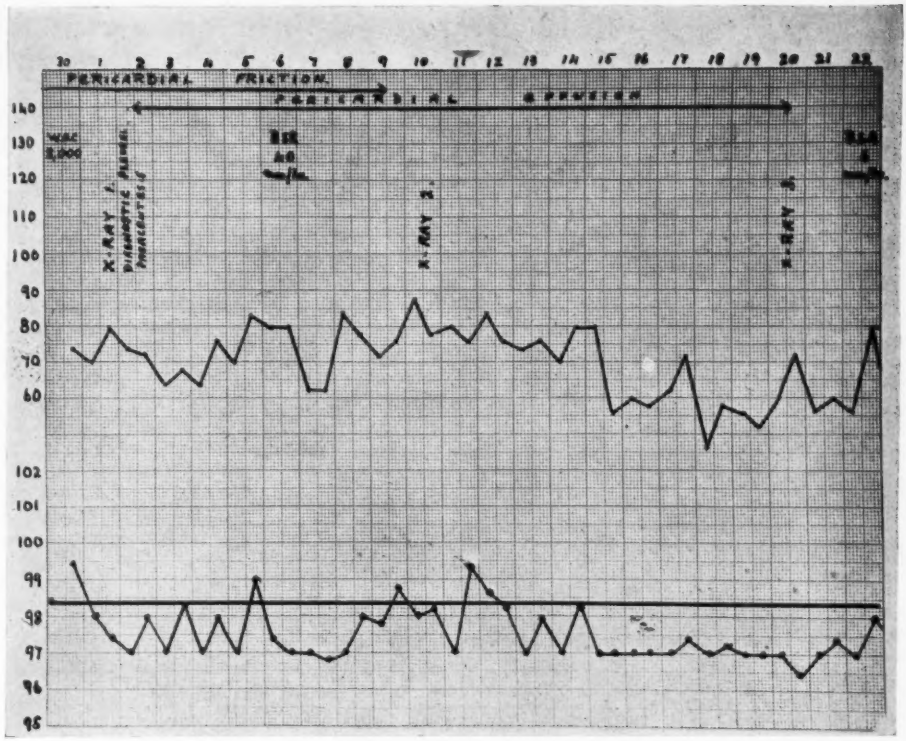


FIG. 11.—Case 3. Temperature and pulse chart.

symptoms of myocardial insufficiency. All writers are in substantial agreement in regarding absence of valvular lesions as an important diagnostic point in the differentiation of tuberculous and rheumatic pericarditis.

Errors in diagnosis might arise from the fact that pericardial thickening can produce exaggeration of the auricular impression on the œsophagus similar to that seen in mitral stenosis (Rubin, 1948); but clinical signs of the valvular lesion are absent. Pericardial effusion, at one stage during its absorption, appears to be capable of producing a similar radiological picture. Thus Case 3, whose first X-ray showed a fairly large pericardial effusion, was reported as a case of mitral stenosis by the radiologist when his second X-ray was examined; there was, however, never any clinical justification for this diagnosis, and the radiological signs that simulated mitral stenosis had vanished completely a month later. Case 4 was also regarded incorrectly as having mitral stenosis by two independent radiologists.

In these circumstances Case 2 of the present series is all the more remarkable, and appears to be unique. This boy had atypical rheumatism at the age of 11; there was no sore throat and pains were confined to the metatarso-phalangeal and hip joints. Three years later he had a second attack identical with the first, but on this occasion endocardial murmurs were recognized in the mitral area. He was referred to the outpatient department after 8 weeks when he had a mid-diastolic mitral murmur with slight prominence of the left auricle and the pulmonary artery radiologically. There was also an active primary Ghon lesion with hilar gland enlargement on the right side, and early secondary infiltration of the base of the right upper lobe. Pericarditis appeared a week later, while he was under observation in hospital; it was not accompanied by any disturbance in temperature or pulse rate, and symptomatically it resembled a tuberculous as opposed to a rheumatic pericarditis; it occurred at a time when there was ample evidence of active tuberculosis (radiological,

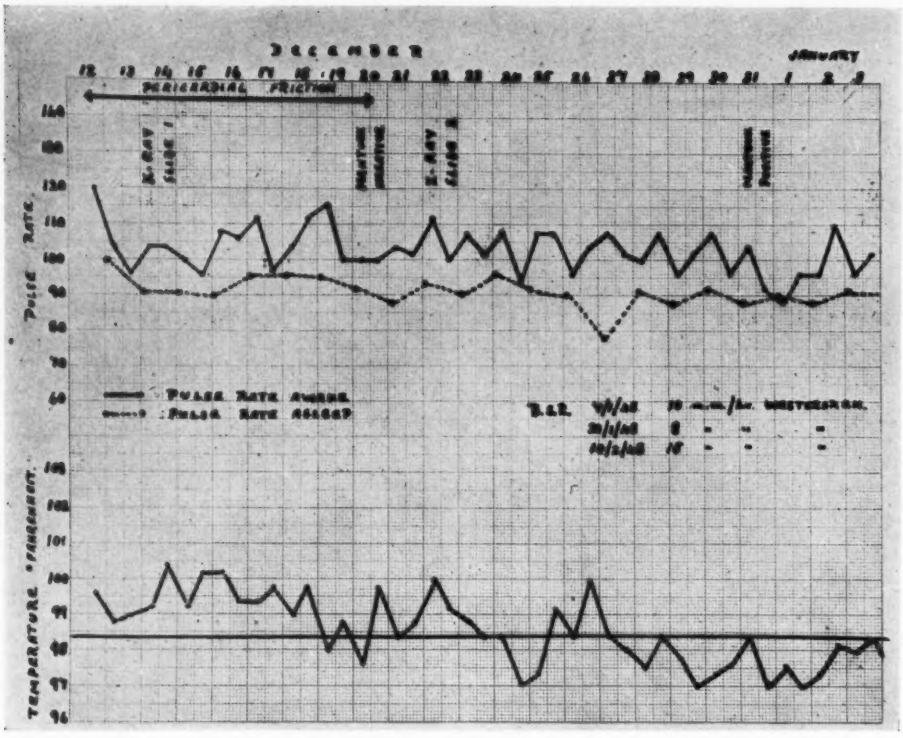


FIG. 12.—Case 1. Temperature and pulse chart.

skin reactions, and lymphocytosis), while there had been no symptoms of active rheumatism for two months. There can be little doubt that the pericarditis was tuberculous. Six months afterwards, the classical presystolic murmur of mitral stenosis was present. The nature of the endocarditis is open to greater doubt. It might be argued that the atypical rheumatism was in reality an example of tuberculous rheumatism, and that the endocarditis was likewise tuberculous. However the first attack of joint pains had occurred three years previously, and the mitral stenosis was more consistent with a 3-year than an 8-week history; whereas the Ghon lesion was undoubtedly his primary tuberculous infection and is unlikely to have been present for more than a few weeks. I believe therefore, that this boy had a rheumatic endocarditis with tuberculous pericarditis secondary to his Ghon lesion.

With the exception of the foregoing case, signs of endocarditis were never found in the series; and none of the remaining patients gave a history of rheumatic manifestations.

## COURSE AND PROGNOSIS

The presence or absence of tubercle bacilli in the pericardial fluid is of the utmost importance in prognosis. Harvey and Whitehill (1937) had a mortality of 83 per cent in their 37 bacteriologically proved cases; Blalock and Levy (1937) also quote a mortality of 83 per cent in a series of 24 bacteriologically proved cases. Contrasting with this, the death rate was only 6 per cent in Harvey and Whitehill's 34 bacteriologically negative cases, which resembled their proved cases in all other respects. None of the latter group showed signs of pericardial constriction on discharge from hospital. Seventeen were followed for periods varying from six months to seven years: one developed signs of adhesive mediastinitis with portal obstruction seven years after having made an apparently complete recovery;

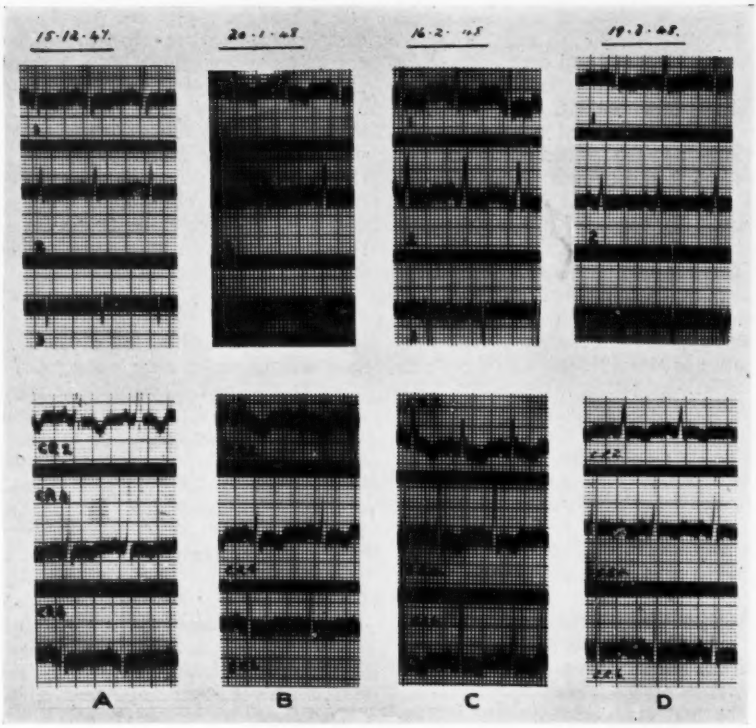


FIG. 13.—Case 1. Serial electrocardiograms. (A) 15/12/47. (B) 20/1/48. (C) 16/2/48. (D) 19/3/48.

two developed tuberculous lesions elsewhere; nine remained well. Ellman (1945) also believes that the disease may become arrested during its course, and that some cases result in a cure at the stage when the effusion has been absorbed.

Two cases in the present series were bacteriologically proved, tubercle bacilli being recovered from the pericardial fluid in one that proved fatal in 12 months, and from the pleural fluid in one whose subsequent fate is unknown. Of the remaining six cases one died, two have apparently recovered (observed for 3 years and 1 year respectively) while three are recent, two of them seemingly recovering.

The various courses which the disease may pursue are well illustrated in the series. In its fully-fledged form it passes through (1) a dry stage, (2) a stage of effusion, (3) a stage of absorption, and (4) a stage of pericardial constriction. Case 4 was observed through all four stages; he was admitted with a dry pericarditis of obscure ætiology and he developed an effusion while under observation; although the effusion absorbed completely before his discharge from hospital he had signs of commencing pericardial constriction in the shape of persistent enlargement of the liver; during the ensuing three months venous distension and œdema appeared and liver enlargement increased. A pericardectomy was followed by a pleural effusion and gave no immediate relief, though there was temporary improvement two months later; nevertheless he relapsed and died six months after the operation. Case 5 illustrates an alternative course that may be followed as the disease becomes chronic. This patient developed a pericardial effusion suddenly while he was convalescent from a tuberculous pleurisy, tubercle bacilli being recovered from the pericardial fluid; the effusion gave rise to venous distension, œdema, and liver enlargement. At first it was possible to aspirate up to 500 ml. from a single puncture; but as time went on the effusion became loculated so that only a few ml. could be obtained from any one puncture. The cardiac failure became

chronic and he died a year after the onset of the effusion.

On the other hand, arrest and cure seem to be possible either before the development of an effusion or after its absorption. Case 6 had pericarditis without demonstrable pericardial effusion on two occasions in 1943–4: in the interval he had a right-sided pleural effusion and co-incidentally with the second attack of pericarditis he had a left-sided pleural effusion. He appears to have made a complete recovery; examination three years after the second attack failed to reveal any abnormality in the heart or lungs. Case 3 had tuberculous pleural and pericardial effusions in May 1947: he made a relatively rapid recovery, returning to work in September 1947; examination in May 1948 showed no evidence of any residual lesion. Of the more recent cases, one (Case 3) seems to have become arrested after a dry stage without effusion, and one (Case 1) shows no cardiac lesion six months after an effusion with myocardial involvement. One case was lost sight of when she was transferred to a tuberculosis hospital in accordance with service regulations. The remaining case is still under observation with a pericardial effusion which has persisted for three months.

The rapidity with which a tuberculous effusion can make its appearance is worthy of mention. Case 3 was seen one evening with pericardial friction: his apex impulse was well-defined and localized, 9 cm. from the midline; the cardiac percussion dullness was not increased; Fig. 7 was obtained on the following forenoon when he was admitted to hospital. The rate of absorption is very variable; in Case 3 the effusion had almost completely absorbed in three weeks (Fig. 8); in Case 5 there was still effusion after eight months despite repeated aspirations.

The features of the chronic constrictive stage have been adequately described by many authors (Pick, 1896; Volhard and Schmieden, 1923; Beck, 1931; White, 1935; Hannesson, 1941; Sellors, 1946), and are outwith the scope of this article. I would agree with Ellman in suggesting that many patients with constrictive pericarditis who give no history of an acute attack have in fact had such an attack, which has been overlooked because of the mildness of the constitutional symptoms. This appears to apply with particular emphasis to those where pericarditis accompanies a primary Ghon lesion, in which subjective symptoms are particularly mild. Thus Case 2 was symptom-free. Case 1 had a pressure cough as his only subjective symptom; I have frequently heard equally severe coughs in children going about the streets, many of whom probably fail to come under medical supervision at all. I have found only one reference to pericarditis complicating an active primary Ghon lesion, and the circumstances are entirely different from those of the present cases. Seligman and Lederer (1940) describe a case in which a secondary hæmolytic streptococcal infection gave rise to an abscess that ruptured into the gland draining a Ghon focus; the gland in turn ruptured into the pericardium producing a suppurative pericarditis. The symptoms in their case were dramatic, simulating either a coronary occlusion or a perforation of an abdominal viscus; the illness was fatal.

## Treatment

Conservative treatment has been employed in most of this series during the acute stage. One (Case 5) who had a persisting pericardial effusion with chronic cardiac tamponade, was repeatedly aspirated in addition to receiving digitalis and mercurial diuretics; he died in 12 months. One other (Case 8), an infant, has had aspiration attempted unsuccessfully; his effusion is slowly absorbing. One case was transferred elsewhere for treatment. The remaining five have been treated conservatively; two are well after 3 years and 1 year respectively; two are convalescent and free from signs of effusion or pericardial thickening after six months; one developed pericardial constriction for which operation was undertaken, but he died six months after the operation.

Treatment by aspiration and air replacement (artificial pneumo-pericardium) is being recommended by some authors; assessment of the value of this procedure will have to await more precise knowledge of the recovery rate in cases treated on purely conservative lines, a rate which would appear to be greater than has hitherto been admitted.

## Summary

The incidence of acute tuberculous pericarditis has been underestimated in this country hitherto. It arises in the early stage of dissemination of tuberculosis: it may be found with the primary Ghon lesion, with an apparently primary tuberculous pleurisy, or in absence of any demonstrable lung or pleural lesions. Infection of the pericardium takes place by spread from the adjacent lymph glands. Constitutional symptoms are mild, sometimes insufficient to lead the patient to seek medical advice; owing to the mildness of symptoms, the diagnosis is apt to be unsuspected. Evidence of myocardial involvement was obtained in one case out of eight.

The disease passes through fibrinous, effusive, and constrictive stages; but it may become arrested and apparently complete recovery may occur

either before the development of an effusion or after its absorption. The effusion may become chronic and loculated giving rise to chronic cardiac tamponade; alternatively absorption of the effusion and apparent recovery may be followed by the development of constrictive pericarditis. The prognosis is extremely poor in cases from whose pericardial effusion tubercle bacilli are isolated; but with a sterile effusion the prognosis is hopeful. Recoveries are by no means uncommon with conservative treatment; care is therefore required in assessing claims for specific therapeutic measures.

## Appendix

*Case* 1. M. aged 11. Health good till November 1947 when harsh cough developed and widespread pericardial friction found; temperature 99° to 101° until admission 14 days later. A sparely built, pale youngster, alert and active, with unimpaired appetite. A harsh metallic pressure cough was his only subjective symptom throughout. Friction persisted over the entire præcordial area for 23 days in all, despite a pericardial effusion of moderate size. Serial cardiograms (Fig. 13) showed evidence of myocardial involvement. Low grade pyrexia (Fig. 12) with slight tachycardia (sleeping pulse 90 to 95). X-rays (Fig. 1 to 3) showed pericardial effusion and an active primary sub-pleural Ghon lesion in left upper lobe with enlargement of left hilar glands. Absorption of the effusion began in 4 weeks and was complete in 6 to 8 weeks; no radiological evidence of pericardial thickening remained. Venous pressure normal throughout, and signs of congestive failure absent. Ghon lesion slow in resolving and still active 6 months after admission. B.S.R. (Westergren) fell from 16 to 8 mm. in 6 weeks, but rose again to 18 mm./hr.

*Case* 2. M. aged 14. Pains in metatarso-phalangeal and hip joints without sore throat or involvement of remaining joints when 11; confined to bed for several weeks. Identical attack September 1947 at age 14; mitral endocardial murmurs recognized. Referred after 8 weeks in bed. A sparely built, pale youngster, but bright, active and cheerful like Case 1, insisting that he felt perfectly well. Mid-diastolic mitral murmur and pulse 110; admitted with diagnosis of rheumatic carditis. Afebrile with sleeping pulse 80 (Fig. 10). X-ray (Fig. 4 to 6) showed active primary sub-pleural Ghon lesion in right upper lobe with hilar gland enlargement and secondary infiltration of base of right upper lobe; slight prominence of pulmonary artery and in oblique view distinct prominence of left auricle consistent with early mitral stenosis.

A week after admission pericardial friction present for four days without disturbance of pulse or temperature. Vollmer patch test positive. Absolute lymphocytosis present (W.B.C. 12400, lymphocytes 44%=5500). No tubercle bacilli on gastric lavage. The Ghon lesion disappeared in between 3 and 4 months. The secondary infiltration extended first, then receded. By April 1948 there remained only a small effusion in lesser fissure with slight pleural thickening; now has a typical presystolic mitral murmur but no signs of pericardial thickening. Serial cardiograms showed no characteristic abnormality.

*Case* 3. M. aged 27. Dysentery and quinsy in Army, but well and category A1 on demobilization February 1946. April 1947, noted attacks of slight præcordial pain and tightness, lasting half an hour at a time, but not preventing work as van driver. After 2 weeks a more severe attack with breathlessness; muffled heart sounds and elevated temperature reported by doctor. Two days later (30/4/47) pain gone, insisted he felt well, but widespread coarse pericardial friction present without cardiac enlargement or displacement of impulse; small pleural effusion present at right base. On admission next day a large pericardial effusion had developed (Fig. 7). There was never venous distension, liver enlargement or œdema. He was virtually afebrile (Fig. 11) and had no tachycardia; bradycardia during convalescence. Despite effusion pericardial friction persisted several days. Pleural fluid contained 1100 cells per cu.mm., 86 per cent lymphocytes; no tubercle bacilli seen in films; cultures sterile. Præcordial pain not severe, recurred once a week later for half an hour; otherwise he had no subjective symptoms. Absorption of pleural and pericardial effusions began within 3 weeks and was complete in 6 weeks. Transient enlargement of the left hilar glands (Fig. 7 and 8) occurred while effusion absorbing; at this stage left auricle and pulmonary artery were prominent, and radiologist reported mitral stenosis: but there was never clinical justification for this diagnosis, and radiological appearances vanished later. Discharged from hospital end of June and resumed work September 1947. On re-examination in May 1948 no abnormality in heart or lungs, and symptom-free.

*Case* 4. M. aged 23. No previous illness, 2 years' active service. Coryza and cough 14 days before onset. On 10/8/41 pain across chest, breathlessness, increased severity of cough and intermittent headache; occasional vomiting. Oedema of legs developed and lasted 5 days but had subsided when admitted to E. M. S. hospital on 27/8/41. Pericardial friction and small pericardial effusion; pulse 100 to 110; B.S.R. 50 mm./hr.; afebrile. Left pleural effusion appeared a fortnight later; X-ray now reported to show " enlargement of heart with mitral configuration "; no endocardial murmurs detected at any time. Cardiogram showed R–T elevation followed by T inversion in all limb leads. Pericardial effusion absorbed a month after admission, but small right-sided pleural effusion developed. By December convalescent, B.S.R. 9 mm./hr., effusions absorbed, but liver enlargement present. Discharged from army and hospital, 17/12/41.

Cough, breathlessness, and œdema soon recurred. Admitted to Victoria Infirmary 21/3/42 with small heart, feeble impulse, distant sounds, enlarged liver, distended neck veins, and venous pressure 24·5 cm. Feeble cardiac pulsations on cardioscopy; prominence of pulmonary

artery and left auricle again noted, and a second radiologist diagnosed "mitral stenosis with pericardial thickening." Pericardial resection performed by Mr. Bruce Dick on 10/4/42 was followed by jaundice, then right pleural effusion; œdema became considerable. Improvement began after a month and he was up after two months. On 6/10/42 he was convalescent and chest radiologically clear. Four days later severe pain in left chest with breathlessness and cyanosis; pleural effusion developed rapidly and he died next day with an effusion of 2300 ml. in left pleural cavity. At autopsy heart was small and there was no mitral stenosis. The excised pericardium showed tuberculous granulation tissue.

*Case* 5. M. aged 43. Admitted to fever hospital in spring 1935 with bilateral pleurisy; pleural fluid and sputum positive for tubercle bacilli. Old apical scar on X-ray but no obvious fresh lesion. After 4 months, effusions absorbed, sputum negative for tubercle bacilli, no active lesion radiologically. Discharge from hospital arranged, but meantime breathlessness developed and large pericardial effusion found; aspirated several times and tubercle bacilli recovered from fluid. Transferred to Victoria Infirmary two months later; afebrile with large pericardial effusion and small pleural effusion on each side; breathless, enlarged liver, ascites, and slight œdema. Pericardium aspirated on two further occasions; no tubercle bacilli found, yet tuberculosis of larynx and palate developed 5 weeks after admission. Paroxysmal auricular fibrillation appeared, and œdema became gross requiring Southey's tubes as well as salyrgan. Oedema cleared but heart size remained unchanged and effusion was now loculated so that only 15 to 30 ml. could be withdrawn from any single puncture. Throat also improved and sputum was negative. Three months after transfer he was up and allowed home prior to entering a sanatorium where he died three months later; the circumstances of his death are not known.

*Case* 6. M. aged 15. History of sore throats periodically but no acute rheumatism. July 1943 fell from bicycle striking chest against pavement, but seemed none the worse after. October 1943 developed præcordial pain and fever; doctor reported cardiac irregularity; was kept in bed 3 weeks and referred a week later. A thin youngster with unhealthy tonsils, "pink and white" complexion, but no clinical signs of heart disease; heart shadow and lung fields normal radiologically; poor voltage QRS in cardiogram; B.S.R. 32 mm./hr. Westergren. Regarded as convalescent from acute pericarditis and sent to E.M.S. hospital.

Improved slowly and B.S.R. dropped to 3 mm./hr. after 10 weeks. Tonsillectomy on 11/2/44 followed by severe hæmorrhage requiring transfusion. On 28/2/44 he had pain in right side of chest with fever (T. 103°), and signs of patchy consolidation followed by a massive effusion; the fever failed to respond to sulphonamides. The pleural fluid contained lymphocytes and polymorphs in equal numbers and was negative on examination for tubercle bacilli. After aspiration resolution was rapid; a trace of fluid remained in right costo-phrenic angle when discharged from hospital on 4/4/44, having gained 15 lb. in weight.

Twelve days after discharge he had slight staining of sputum with tiredness, flatulence, and a little pallor; but no fresh clinical or radiological findings in heart or lungs, and weight was maintained. A month later again tired, listless, off his food, breathless, and slight cough; fainted; when next seen on 8/6/44 pericardial friction and left pleural effusion found. Readmitted for 5 weeks.

Well since, though there is mild effort syndrome and occasional tendency to faint. On 30/10/47 no abnormality in heart or lungs clinically; cardiogram physiological; on X-ray, heart shadow a little displaced to left but otherwise normal, traces of former pleurisy at left base without any remaining fluid, and slight residual fibrosis on right side. Tuberculous ætiology in this case is unproved but seems highly probable.

*Case* 7. F. aged 20. A girl, serving in the W.A.A.F., previously healthy, admitted with acute pleurisy; febrile and flushed with tachycardia and signs of large right-sided pleural effusion. Pericardial friction noted on admission, and pericardial effusion appeared a day or two later. Pleural exudate was lymphocytic and positive for tubercle bacilli. Transferred to a service tuberculosis hospital and further course is unknown.

*Case* 8. M. aged 18 months. Acute poliomyelitis at 13 months, September 1947, involving left arm and leg. Admitted orthopædic unit 29/10/47. Leg recovered during next two months but arm remained paralysed and in abduction plaster splint. Cough with signs of bronchitis noted from time to time.

February 1948, listless and refused food; large pericardial effusion found on X-ray; no abnormality in lung fields. Transferred to medical ward under Dr. A. M. Scott. A well nourished youngster, pale, but mentally alert; heart sounds distant, no friction heard; liver enlarged, two finger-breadths below costal margin on admission, down to umbilicus six weeks later. There was no œdema, breathlessness, nor distension of neck veins. He was virtually afebrile, a temperature of 100° having been recorded on two occasions and 99° once; pulse 110 to 120. Cardiogram shows low T waves in leads I and II with inverted T III, and left axial deviation. Mantoux test negative on 1/3/48 and 26/4/48. An attempt to aspirate pericardium on 5/4/48 yielded blood only. X-ray appearances remained unaltered until 25/5/48 when commencing absorption of effusion was noted; it has now reappeared (July 1948) with œdema of arms and legs.

He is still under observation. Tuberculosis is suspected as the cause, but is as yet unproved.

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# ABSTRACTS OF CARDIOLOGY

**The Coronary Circulation.** O. D. RATNOFF and M. PLOTZ. *Medicine, Baltimore*, **25**, 285–342, Sept., 1946.

A review of previous work on the coronary circulation, e.g. various factors affecting coronary flow, the nervous control and the pharmacology of the coronary circulation, and the coronary blood flow in disease. Because of the lack of methods suitable for application to man or even to intact animals, the coronary circulation has been studied mainly on isolated animal hearts or heart-lung preparations. The results so far are therefore mainly of academic interest. The authors point out that differences in anatomy and physiology of the coronary vessels among various species as well as among individuals of the same species have not hitherto been adequately stressed. Discretion must be used in applying information derived from artificial preparations to the intact animal, from species to species and even among individuals of the same species. Conclusions with regard to human coronary physiology have been drawn freely from animal experiment. *R. T. Grant*

**Cardiac Murmurs in Infancy.** N. COX. *Brit. med. J.*, **1**, 148–150, Jan. 24, 1948.

In a preliminary report on the significance of cardiac murmurs in infancy the author records observations on 630 ostensibly healthy children examined at the ages of about 6 weeks, 3, 6, 9, and 12 months, and thereafter at 6-monthly intervals, most of them for about 3 years. Regular 6-monthly radiographic examinations were made in all children. Thirty-two (5%) had murmurs which were classified according to the age when first noticed and are described as transient, temporary, or persistent. No diastolic murmurs were observed. Of the 32 murmurs 23 were not detected until after the child was 9 months old. There was a relatively greater number of transient murmurs among infants observed in the early weeks of life. Six out of 8 murmurs appearing at the age of 18 months remained permanent. With the exception of 2 cases, no associated symptoms or signs could be observed. No correlation between clinical findings and radiographic appearances could be established. The results are as yet too few to allow any conclusions to be drawn. *H. Herlinger*

**Syndrome of Aberrant Right Subclavian Artery with Patent Ductus Arteriosus.** H. P. BREAN and E. B. D. NEUHAUSER. *Amer. J. Roentgenol.*, **58**, 708–716, Dec., 1947.

Aberrant right subclavian artery is the most common congenital anomaly of the aortic arch. In such cases the right subclavian artery emerges from the extreme left side of the aortic arch, posteriorly and to the left of the origin of the left subclavian. The aberrant vessel runs obliquely upwards and to the right, usually passing behind the œsophagus as it crosses the midline. This anomaly is often accompanied by other congenital anomalies of the large vessels at the base of the heart. Five cases of this syndrome are reported; 3 were accompanied by a patent ductus arteriosus, 1 by a tetralogy of Fallot, and 1 by a cardiac anomaly of uncertain nature. The radiographic features of anomalous right subclavian artery and of patent ductus arteriosus are reviewed and the value of a barium "swallow" during the radioscopic examination of the heart is stressed. *A. Orley*

**The Syndrome of Thrombotic Obliteration of the Aortic Bifurcation.** R. LERICHE and A. MOREL. *Ann. Surg.*, **127**, 193–206, Feb., 1948.

Thrombotic obliteration of the aortic bifurcation must not be confused with the well-known "saddle embolism." Important features are said to be (1) sexual disturbances —poor erection and ultimate impotence; (2) intermittent claudication due to fatigue in the limbs rather than to actual pain; (3) symmetrical atrophy and pallor of both legs; (4) no pulses at groins or below; (5) no oscillometric readings in lower limbs; (6) ultimately gangrene. Aortography may be used to confirm the diagnosis.

Treatment is aimed at: (1) Excision of the thrombotic segment to prevent spread of clot and production of vasoconstrictor impulses. (2) Improvement of collateral circulation in the lumbar region by high lumbar ganglionectomy. In good-risk patients this should be accomplished by excision of the thrombosed aortic segment and obliterated branches through a left iliac incision. Left lumbar ganglionectomy is performed at the same time, and upper right lumbar ganglionectomy at a later stage. If the condition is due to thromboangiitis obliterans unilateral adrenalectomy is advised. In poor-risk patients ganglionectomy should be performed first and aortectomy later if the patient's condition will allow. *J. B. Kinmonth*

**A New Anomaly of the Aorta: Left Aortic Arch with Right Descending Aorta.** R. N. PAUL. *J. Pediat.*, **32**, 19–29, Jan., 1948.

From Johns Hopkins University, the author reviews the common anomalies of the aorta, classifying them and commenting on some, with particular reference to 2 cases of a previously unreported anomaly, that of left aortic arch with right descending aorta.

He differentiates between right aortic arch with right descending aorta and right aortic arch with retro-œsophageal aorta (right and left). He describes the

double aortic arch and comments on the common variations in the subclavian artery. He then presents, in great detail, his cases of the new anomaly. Each patient had been considered to have the tetralogy of Fallot, and both were candidates for the Blalock-Taussig operation.

The first, a 7-year-old white female gave a history of cyanosis from early infancy and extreme dyspnœa on the slightest activity. Radioscopy of the œsophagus showed: (1) indentation of the left margin by the aortic arch, establishing the fact that a left-sided arch existed; (2) absence of any vascular or cardiac shadow to the left of the barium column between the aortic knuckle and the upper left border of the heart; (3) descent of the œsophagus far to the left of the midline. At operation the course of the aorta was demonstrated, but no anastomosis was performed as the pressure in the pulmonary artery was considered too great. The second, an 11-year-old white male, had had cyanosis from the age of 1. Radiographs had the same characteristics as in Case 1. An anastomosis was performed, but the course of the aorta could not be clearly demonstrated, though by inference this case appears to have also been one of left aortic arch with a right descending aorta.

*Morag L. Insley*

**The Surgical Relief of Severe Angina Pectoris. Methods Employed and End Results in 83 Patients.** J. C. WHITE and E. F. BLAND. *Medicine, Baltimore*, **27**, 1–42, Feb., 1948.

This paper reviews the methods of interrupting the afferent pain fibres from the heart in cases of angina pectoris, and presents the clinical results in 83 cases.

The methods available are: (1) Paravertebral injection of procaine followed by 95% ethyl alcohol around the upper four thoracic sympathetic ganglia. (2) Sympathetic ganglionectomy. The stellate and upper three thoracic ganglia are resected extrapleurally on one side only, the other side being dealt with later if necessary. (3) Posterior rhizotomy. The upper four thoracic posterior spinal nerve roots are sectioned on both sides. This is undoubtedly the most effective way of cutting off all afferent impulses from the heart at one operation, but it is a major and time-consuming operation and carries the risk of ischæmic transverse myelitis.

Surgical treatment should be reserved for those whose pain, after adequate observation, cannot be controlled effectively on a medical regime, and especially for angina decubitus. Patients with good cardiac reserve and bilateral pain are suitable for laminectomy and thoracic root section. For patients in whom the risk is slightly greater, with unilateral pain, thoracic ganglionectomy is preferred. Where the risk is very great, paravertebral block with alcohol is the only possible routine.

Clinical results in 75 patients treated with paravertebral alcohol block are reviewed; 56% were completely relieved of pain; in 21% partial relief only was obtained; 8% died as a direct result; 10% developed severe intercostal neuralgia. Of the group in whom initial results were good there was recurrence of pain due to recovery of nerve conduction in 19% after periods varying from 2 months to 5 years. Eight cases have been treated by left-sided thoracic ganglionectomy. Complete relief of pain on the denervated side was obtained in all. There was 1 death (from empyema). In 1 case there was slight recurrence of pain after 6 years, due to nerve regeneration. One patient was treated by posterior root section with complete relief.

Although the number of cases is not large enough to be statistically significant, results show that prognosis as regards life in cases of severe angina is not altered by cardiac denervation. In angina decubitus with loss of sleep, worry, and threatened drug addiction relief from pain and anxiety has been effective. After complete division of all afferent nerves the place of anginal pain as a warning signal is taken by constriction or oppression referred to the suprasternal notch, and painless dyspnœa. Occasionally a pain referred to the jaw of the same side becomes evident; the mechanism of this is not understood. The authors consider that thoracic ganglionectomy should be the operation of choice whenever possible.

*F. B. Cockett*

**The Efficiency of Maintenance Doses of Digitalis in Preventing the Recurrence of Congestive Heart Failure.** J. S. LAUDE and S. B. CARTER. *Ann. intern. Med.*, **27**, 923–928, Dec., 1947.

Doubt is cast upon the value of estimations of the vital capacity, circulation time, venous pressure, and body weight as indications of the extent of cardiac reserve in patients who have been safely brought through a phase of congestive heart failure. It would appear that a careful evaluation of symptoms and clinical signs is a better guide. Patients who have been relieved of congestive heart failure, even with regular sinus rhythm, should always be placed upon maintenance courses of digitalis leaf. The present study of 104 patients shows that patients given such maintenance doses are much less likely to relapse than are similar patients not given maintenance doses. A suggested maintenance dose is 0·1 to 0·3 g. of digitalis leaf daily.

*G. F. Walker*

**Lanatoside C in the Treatment of Persistent Paroxysmal Auricular Tachycardia.** A. S. WEISBERGER and H. FEIL. *Amer. Heart J.*, **34**, 871–877, Dec., 1947.

In a series of cases of persistent paroxysmal auricular tachycardia, the tachycardia ceased abruptly within 40 minutes after the intravenous injection of 0·8 mg. "lanatoside C." No toxic reactions or undesirable side-effects occurred.

*R. T. Grant*

**Hyperpiesis with Atheromatous Obstruction of the Renal Arteries.** G. T. COOK and R. S. B. PEARSON. *J. Path. Bact.*, **58**, 564–567, Sept., 1946.

This is a brief single case report of a 29-year-old man with developmental anomalies and hypoplasia of the vascular system, who showed severe hypertension (300/150 mm.) and cardiac failure (heart weighing 560 g.), although the histological changes in the kidneys were very slight. The right subclavian and right common carotid arteries showed recanalization following complete obliteration. No changes were noted in the fundi, and the Wassermann reaction was negative. Comparison is made with the results of experimental occlusion of renal arteries.

*A. C. Lendrum*

**Differential Diagnosis of Retrocardiac Shadows.** S. S. NEMEC. *Radiology*, **50**, 174–183, Feb., 1948.

The most obscure thoracic lesions are those in the retrocardiac space where there is ample room and where consequently a lesion may reach considerable size before it causes clinical signs of compression. Retrocardiac shadows may be produced by lesions of the œsophagus, stomach, lungs, spine, paravertebral tissues, and aorta. The author describes a diagnostic sign consisting of multiple convex lines within the cardiac shadow, indicating the presence of the stomach in the retrocardiac region. He also describes a simple double-contrast method (gas-distension of stomach and barium filling of œsophagus) for the study of the cardio-œsophageal junction and of the relation of the lower third of the œsophagus to the stomach. Finally, the author asserts that a diagnosis of asymptomatic retrocardiac lesions may be made from observation of abnormal retrocardiac shadows in routine radiographs of the chest. *A. Orley*

**Penicillin in Subacute Bacterial Endocarditis. Report to the Medical Research Council on 269 Patients Treated in 14 Centres Appointed by the Penicillin Clinical Trials Committee.** R. V. CHRISTIE. *Brit. med. J.*, **2**, 1–4, Jan. 3, 1948.

Penicillin can now be expected to control the infection in about 90% of patients suffering from subacute bacterial endocarditis, but about 35% of these die of heart failure or other complications.

Early schemes of treatment showed that, at least with smaller courses of penicillin, such as a total of 5 million units, the duration of treatment was of greater importance than the amount of penicillin given daily. For this reason, in a later group of 158 previously untreated patients, the duration of treatment was established at 28 days—of 17 who received 100,000 units daily, 7 relapsed or died of infection; of 83 receiving 250,000 units daily 13 relapsed; and of 58 who received 500,000 units daily 4 relapsed. Only occasionally can failure be explained as due to a highly resistant organism, but when the coefficient of resistance of the organism is more than about 8, it is important to give larger doses, possibly over an extended period.

Of 42 patients who had relapsed after a long course of treatment, 59% again relapsed or died infected. Acquired resistance to penicillin is seldom an important factor in this high incidence of failure; it may be that these patients belong to a naturally resistant group or that the infecting organisms are buried in avascular tissue. A course of 2 million units a day for 8 weeks is suggested for patients who have relapsed. Relapses seldom occur more than a month after treatment, but a small risk remains even after a year or more of good health. It is sound practice to remove foci of infection, particularly in the teeth, while the patient is receiving penicillin. The majority of deaths occurred either during treatment or soon after its completion. Heart failure caused 40% of the deaths and was a major factor in another 16%. At necropsy apparently living organisms were found in the heart valves of one-third of those whose infection, according to the usual clinical criteria, had been controlled.

In prognosis the nature of the initial endocardial lesion is of slight importance; the highest death rate was found in combined aortic and mitral lesions, and the lowest in septal defects. Evidence of heart failure is much more significant; in a group of patients where this was absent 36·4% died; of those with moderate failure 75% died, and of those who had severe heart failure when treatment was started all died. Of those recently infected 34% died, while of those infected for 6 months or more over 60% died. The state of nutrition is another significant factor; this emphasizes the need for an adequate calorie intake as with other prolonged fevers. Only in the group over 50 should age influence the prognosis. *T. Semple*

**Further Observations of Prothrombin Determinations and Vitamin K Therapy in Acute Coronary Occlusions.** H. MCG. DOLES. *Sth. med. J.*, **40**, 965–973, Dec., 1947.

The author carried out frequently repeated determinations of prothrombin level in 64 cases of acute coronary occlusion and in 657 controls free from coronary disease, and found significant hypoprothrombinæmia in the coronary cases—that is, with a level below 70% of normal. This stage is considered as heralding an episode of bleeding in general, and of coronary subintimal hæmorrhage in particular, thus precipitating coronary thrombosis. The frequent relation between gall-bladder disease and coronary disease, with the known hypoprothrombinæmia in the former, is commented upon. Cases of coronary occlusion were treated with intramuscular doses of vitamin K (50 to 72 mg. every 6 to 8 hours) until the prothrombin level reached 100% of normal. Rapid cessation of pain under this regime was observed. In some cases prothrombin levels of 100% of normal were encountered immediately after the onset of the coronary occlusion, but in these cases a fall of from 30 to 50% below the first reading was encountered 6 to 8 hours later. The initial high level in these cases is regarded as a compensatory mechanism that becomes exhausted within a few hours. A mortality rate of 4% in this series is arrived at by excluding 8 patients dying before adequate treatment could be given and 3 whose treatment was considered inadequate. The author makes no attempt to reconcile his claims with those of workers who use heparin and dicoumarol. *G. Schoenewald*

**Complications of Blood-letting in Hypertension.** L. WALTERSKIRCHEN. *Wien. klin. Wschr.*, **59**, 822–824, Dec. 19, 1947.

In recent years venesection has been less used in the treatment of hypertension, and is now condemned by many authorities as dangerous. Angiospasm, angiospastic hemiplegia, and collapse are some of its dangers. Two cases are reported in which electrocardiographic findings are given before and after venesection in hypertensive patients with anginal symptoms. From these findings and from experimental data it is concluded that venesection may lead to acute coronary insufficiency by causing a sudden fall in aortic pressure. The prognosis in these cases is otherwise better than in patients who have anginal symptoms with a low blood pressure.

*Harold Jarvis*

**Tetraethyl Ammonium Bromide in Hypertension and Hypertensive Heart-failure.** G. W. HAYWARD. *Lancet*, 1, 18–20, Jan. 3, 1948.

Tetraethylammonium bromide (T.E.A.B.) was given by slow intravenous injection in a 10% solution in doses of 0·3 to 0·5 g. (4 to 6 mg. per kilo body weight). The effects were compared with those of "sodium amytal" in a preliminary group of 30 patients with essential hypertension. With T.E.A.B. the average fall in blood pressure was 58 mm. Hg systolic and 28 mm. diastolic; with sodium amytal it was 74 mm. and 35 mm., respectively. T.E.A.B. produced no ill effects, and its action was transient. Ten patients were operated on (removal of sympathetic chain and ganglia from D6 to L1), and in 7 cases pre-operative depression with T.E.A.B. agreed to within 10 mm. Hg with diastolic depression recorded 2 weeks after completion of the second stage of the operation. Sodium amytal gave similar agreement in 9 cases. Four patients were further investigated after operation, and in each case T.E.A.B. gave a level lower than that in the pre-operative test. Six patients with hypertensive heart failure were treated with T.E.A.B., some by intramuscular injection of 1 g. All patients showed temporary subjective improvement, often for some hours. Electrocardiograms showed no change, and no cardiac pain or infarction has been reported after injections. The speed and safety of the test with T.E.A.B. is not associated with an accuracy equal to that of the sodium amytal test, and this is attributed to the fact that the former drug produces only partial ganglionic paralysis, as indicated by its increased action after sympathectomy. The beneficial effects of T.E.A.B. in hypertensive heart failure are attributed to decrease in pulmonary congestion similar to that following venesection, and last 2 to 8 hours after intramuscular injection. Since venous pressure falls in hypertensive cases to an equal extent with or without failure, the fall is considered to be due to a lowering of peripheral resistance. The drug is thus of use in the emergency treatment of acute left ventricular failure or paroxysmal nocturnal dyspnœa. In the treatment of chronic congestive failure the fall in urinary output which results from the lowering of blood pressure makes its use inadvisable at present. *W. A. Bourne*

**Paroxysmal Diaphragmatic Flutter with Symptoms Suggesting Coronary Thrombosis.** F. H. MOORE and C. SCHOFF. *Amer. Heart J.*, **34**, 889–893, Dec., 1947.

A brief description is given of a patient suffering from severe anginal pain associated with flutter of the diaphragm. On præcordial auscultation a loud booming noise was heard, its frequency being about 200 a minute; palpation of the lower thorax conveyed a marked throbbing sensation synchronous with the sounds. On radiological examination both leaves of the diaphragm were seen to be fluttering rapidly, in time with the palpable impulses and the sounds. Morphine, gr. ¼ (16 mg.), relieved the anginal pain but the flutter continued; this ceased several hours later and did not recur. The patient, a psychopath, was recognized as the individual who, under various aliases, had been admitted to other hospitals with the same complaint from time to time since 1936. References are given to 4 other published accounts of the case. *R. T. Grant*

**Radiological Differentiation between Pericardial Effusion and Cardiac Dilatation.** J. ARENDT. *Radiology*, **50**, 44–51, Jan., 1948.

The pericardial space in health is fairly lax and has an appreciable capacity for accumulating fluid without presenting the characteristic contours. When accumulation is rapid, the cardiac contours are obliterated and the spherical form with a short pedicle is seen. Slow accumulation of fluid gives time for some accommodation of the pericardium and then differentiation from cardiac dilatation may not be easy. The author has found Valsalva's test followed by forced expiration a useful means of differentiation. The dilated heart responds, first by becoming smaller, and then by notable increase in size with enlarged amplitude of pulsation. In the presence of a pericardial effusion there is little or no variation in size or visible behaviour. The bifurcation of the trachea may be a further useful index. In cases of dilatation, especially when the left auricle is enlarged, the bifurcation angle is widened from the normal 75 degrees to over 100 degrees. When enlargement of the heart shadow is due to fluid there is only slight widening, if any. A further useful point is that, in the latter condition, fluid tends to obscure the carina, while in cardiac dilatation the bifurcation is usually clearly visible. Pericarditis with effusion may displace the barium-filled œsophagus to a disproportionately slight extent and the curvature is wide and smooth. The dilated heart is likely to displace and impress the œsophagus more definitely, the impression being sharper and of a more localized nature. Kymography may give more precise evidence of the diminution in pulsation occasioned by fluid. *A. M. Rackow*

**Roentgen Demonstration of Calcifications in the Interventricular Septum in Cases of Heart Block.** F. WINDHOLZ and C. GRAYSON. *Amer. J. Roentgenol.*, **58**, 411–421, Oct., 1947.

Calcification in the membranous septum of the heart, sometimes associated with heart block, can be demonstrated radiologically. The authors' views are based on 61 patients who showed radiological calcification in the mitral or aortic rings; of these, 12 had some degree of heart block. In the left oblique view the mitral ring is projected in its most open form and the posterior limb, when calcified, clearly shown. The authors regard as evidence of probable septal involvement: (1) caudal extension of aortic ring calcification (see in 2 cases with heart block); (2) complete calcification of the mitral ring (see in 8 cases with heart block); (3) incomplete calcification of the mitral ring with an aggression of calcium at the medial end of the posterior limb (see in 2 cases of heart block). The last type was observed also in cases without heart block, and is considered to be a less specific sign than the first two. *A. M. Rackow*

**Chronic Auricular Fibrillation. Its Treatment with Quinidine Sulfate.** R. L. McMillan and C. R. Welfare. *J. Amer. med. Ass.*, **135**, 1132–1136, Dec. 27, 1947.

This paper, based on a study of 50 unselected cases, is a plea for a wider use of quinidine sulphate. In the authors' view the only definite contraindications to its use are severe conduction defects, subacute bacterial endocarditis, and angina pectoris, previously relieved by the onset of fibrillation. All their patients were admitted to hospital and complete digitalization was effected before quinidine therapy was instituted. An average increase of 270 ml. in vital capacity followed return to normal rhythm in 18 patients, and an average decrease in venous pressure of 20 mm. of saline solution in 13 patients. The average decrease in circulatory time in 14 patients was 6·7 seconds. After a test dose of 0·2 g., quinidine was given every 4 hours day and night. The dosage began at 0·5 g. and was increased by 0·2 g. every 12 hours until normal sinus rhythm occurred, toxic symptoms appeared, or 0·8 g. was reached. Further increase of dosage was slow, usually by 0·1 g. per dose every 24 hours. After reversion to normal rhythm quinidine was given in the same dosage at 6-hour instead of 4-hour intervals.

In 35 patients for whom a satisfactory maintenance dosage was worked out, the average amount necessary for reversion was 0·45 g. every 4 hours, and the average maintenance dose was 0·28 g. every 6 hours. Sinus rhythm was restored in 44 patients, of whom 14 had maintained normal rhythm for from 4 months to 2 years, half of them being on maintenance doses of quinidine. In the 5 cases of thyrotoxic heart disease treatment was unsuccessful until hyperthyroidism had been corrected. Apart from the 2 patients under 20, best results were obtained in the 40-to-49-year age group, in rheumatic heart disease, and in those in whom the adequate 4-hourly dose did not exceed 0·3 g. It is claimed that restoration of normal rhythm may do much to prevent the development of a cardiac neurosis. Eleven of the 50 patients studied died, during the time of observation which was apparently a period of 2 years.

The authors consider that this study indicates that much larger doses of quinidine sulphate may safely be administered than has been thought practicable. [It is obvious (as in the 3 fatal cases quoted) that the range of patients treated was much wider than that considered suitable by most British cardiologists.]

*Donald Hall*

**Blood Volume in Congenital Heart Disease.** D. E. Cassels and M. Morse. *J. Pediat.*, **31**, 485–495, Nov., 1947.

A shunt occurs in cases of auricular and ventricular septal defect, and in patency of the ductus arteriosus. These communications should have an effect on the circulation similar to that of a peripheral fistula, initiating an accessory circulation that requires a certain volume of blood to fill it. Theoretically this blood can be supplied by: (1) reduction of blood flow distal to the fistula, (2) increase in total blood volume, or (3) both.

Several investigators have found an increased blood volume in patients with arteriovenous fistulæ. The present authors contribute blood volume estimations (by the blue-dye method of Gibson and Evans) on normal children and patients with congenital heart disease. The latter were studied in three general groups: (1) patent ductus arteriosus; (2) acyanotic congenital heart disease, not including coarctation of the aorta or aortic stenosis, since abnormal shunts do not occur in these cases; (3) cyanotic congenital heart disease of varying type. In 13 patients with uncomplicated patent ductus the blood volume was elevated—considerably in 4, while 1 had a blood volume below the mean normal. In 7 of 8 instances where the blood volume was estimated before and after ligation there was a decrease in blood volume following surgery. The blood volume in acyanotic intracardiac arteriovenous shunts was elevated by a smaller amount. It is tempting to attribute this to the smaller volume of blood passing through the fistula, since in intracardiac shunts the flow is probably intermittent, while in extracardiac shunt the flow is continuous, though uneven. In the cyanotic type of congenital heart disease blood volume was greatly increased, the increased red cell volume being usually associated with diminished plasma volume. This resulted in a rise in viscosity.

*M. Baber*